JOURNAL
OF THE
AMERICAN VETERINARY MEDICAL ASSOCIATION

May, 1939

Published by
AMERICAN VETERINARY MEDICAL ASSOCIATION
221 N. LA SALLE STREET
CHICAGO, ILL., U.S.A.

Volume XCIV N. S. Volume 47, No. 5

Milk Fever. Injection being made with Simplex Outfit. Vein distended with tourniquet, and site of injection swabbed with Malgreen. (Illustration courtesy of Dr. D. E. Trump, Utica, Neb.)

The Problem

MILK FEVER

The Answer

Norden Certified

NORCALCINATE 24%

The full dose calcium gluconate. Standard milk fever specific. Ready for any calcium deficiency. NON-PRECIPITATING.

Case of 6-350 cc ..... $3.80 12-100 cc $2.65

NORCALCINATE with DEXTROSE

500 cc is equal to 500 cc Norcalcinate 24% plus 100 grams of dextrose.

Case of 6-500 cc ..$4.50 12-100 cc ..$2.85

NORCALCIPHOS

Calcium gluconate and inorganic phosphate in same quantity and ratio as calcium and phosphorus in blood stream of 1000 lb. cow. Helps prevent relapses.

6-300 cc $4.25 With Dextrose 6-500 cc $5.00

NORDEN SIMPLEX INTRAVENOUS OUTFIT

ALL rubber, pure gum. No metal to corrode. No valves to break. Patented flutter valve—lets in air, holds in fluid. Recommended by veterinarians the world over, for efficiency, simplicity. $1.25

Ask for free booklet on "Calcium Therapy" by Dr. Frank Breed

NORDEN LABORATORIES

LINCOLN "The Mark of Quality" **NEBRASKA**

Official Organ

of the

American Veterinary Medical Association

CONTENTS—

$4.00 per annum. Foreign $5.00; Canada $4.50 Single Copies 40 cts. in U. S.

Published monthly at 221 N. La Salle St., Chicago, Ill. Entered as Second Class Matter, August 19, 1932, at the Post Office in Chicago, Illinois, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103. Act of February 28, 1925, authorized August 10, 1932. Address all correspondence to American Veterinary Medical Association.

Journal
of the
American Veterinary Medical Association

(Registered U. S. Patent Office)

221 N. La Salle St., Chicago, Ill.

Manuscripts are acknowledged on the same day they are received. Reprints should be ordered in advance. Prices will be quoted upon application.

Please send prompt notice of change of address, giving both old and new. Advise whether the change is temporary or permanent.

Vol. XCIV, N. S. Vol. 47 MAY, 1939 No. 5

Infectious Equine Encephalomyelitis

By JAMES FARQUHARSON, D. V. M.
Colorado State College, Fort Collins, Colo.

COLORADO has experienced a number of outbreaks of infectious equine encephalomyelitis. Without a doubt, we had two well known outbreaks of this disease within the last 30 years previous to the discovery of the causative agent in 1930 by Meyer, Haring and Howitt of California. The first appeared in 1912, and was confined to the Arkansas valley in the southeastern part of the state. In 1919 it reappeared in the Arkansas valley and along the Platte valley in the northeastern part of the state—widely separated areas. At that time it was known by a number of ambiguous terms, such as forage poisoning, botulism, Kansas horse plague, and other terms too numerous to mention.

—*After Cox, Philip, Marsh and Kilpatrick, 1938.*

Encephalomyelitis, sleepy type, showing animal just prior to falling.

In 1932 it appeared in widely scattered areas throughout the state, principally along the river valleys. At that time it was proven by Newsom and Cross, of Colorado, to be caused by a filtrable virus, the same as that found by Meyer, Haring and Howitt,[1] of California, and Records and Vawter,[2] of Nevada. It has reappeared every year except 1934, when the entire state seemed to escape. Paradoxically, not a single case was reported during that year. We have therefore, experienced this disease six years out of seven.

The unusual thing about the disease in Colorado is that it continues to appear in the same districts year after year, with a considerable number of resident animals involved in each seasonal epizoötic. In the Poudre River valley it is estimated that we have a 25 per cent transient horse population, which might account for the disease

appearing in newly imported, susceptible animals. This does not explain the situation, however, because many animals that have been in the same place for years escape for one, two or more years, and then succumb to the disease. We have also observed animals that have contracted the disease on successive years. In this respect, our experience does not concur with our expectations, nor with the opinion expressed by others that an epizoötic of this nature should confer an immunity on the horse population of the districts affected.

The paramount factors to be considered in the diagnosis of encephalomyelitis are: first, epizoötiology; second, seasonal occurrence, and third, histopathological findings. sporadic or epizoötic. In our experience, infectious encephalomyelitis usually starts about August 10 to 15, reaching its height during the first and second weeks of September, to terminate with the first killing frost, about October 20 to November 1. In 1938, however, the disease appeared about a month earlier than in previous years, the first case being observed on July 7. Moreover, the disease made its appearance for the first time in the mountainous districts. Previously, we had surmised this area to be rather secure from the spread of this disease. The 1938 epizoötic not only exceeded the number of cases seen in previous years, but the causative virus was more virulent.

—*After Cox, Philip, Marsh and Kilpatrick, 1938.*

Encephalomyelitis, showing dirt-eating symptoms.

The disease, being seasonal, undoubtedly requires an insect vector for its transmission. When frost destroys the carriers, the disease soon ceases. This type of disease may suddenly appear as an epizoötic and spread rapidly; or, in certain localities, it may be more or less sporadic the first year, to reappear at the same time the following year as a veritable epizoötic. This has been true in our experience in Colorado. Therefore, the epizoötic nature and seasonal occurrence make diagnosis a simple matter, once the disease has made itself evident.

The virulence of the disease changes during the season. Some years it appears to be extremely virulent early in the season, and the majority of the cases seen are either acute or hyperacute, with a high death rate. Later the symptoms become much milder in appearance and the mortality drops. This may be due to two factors. Either the more susceptible animals are the first to succumb or the virulence of the virus is greater and gradually subsides. In other seasons it may appear, mildly at first, gaining virulence in the middle of the season, to subside gradually.

The clinical course of the disease may be conveniently divided into: first, the incubative period; second, the lethargic or paralytic stage, and third, the convalescent stage. The incubative state escapes notice unless temperatures are carefully recorded several times daily. There is evidence that many subclinical cases terminate at the end of the incubative period and show little or no evidence of the paralytic symptoms. Others follow a regular course lasting from three or four days to seven or ten days. Neurasthenia or complete prostration may develop within the first three days of the disease.

SYMPTOMS

The onset of symptoms is quite inconstant, being influenced somewhat by the extent and dissemination of the pathological process, as well as by the apparent affinity of the virus to affect certain centers early in the course of the disease. The symptoms also depend upon the individual susceptibility of certain animals to contract the disease in an acute, subacute, or subclinical form.

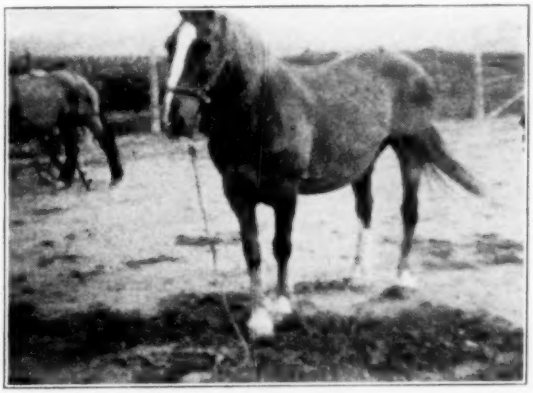
What the animal has experienced until the objective signs become noticeable is always

a question. Most of the cases are well established before the veterinarian sees them. At least, there is sufficient change from normal in the attitude of the animal that it becomes noticeable to the owner, who is not well trained in the powers of accurate observation. Therefore, some of the earliest symptoms are seldom seen by the clinician. The onset must be more or less insidious in such cases. Many descriptions on this subject undoubtedly offer variations because of the time element in observation.

Infectious equine encephalomyelitis, as observed generally, can be classified as a lethargic type of encephalitis. Although in some instances initial irritability is seen, the usual syndrome is that of a progressive disturbance of consciousness, characterized by dullness, lethargy, and lassitude. There is constantly an initial rise of temperature, ranging from 103 to 106°. Low temperatures should generally be interpreted as being on the decline, although it is not unreasonable to assume that a resistant animal or a weak virus may not cause an extremely high temperature.

The temperature drops rapidly to normal or almost normal after the second day. The pulse is slow, soft and full at first, later subject to variation. The respiration is not greatly altered and is of no diagnostic value. The visible mucous membranes are slightly to markedly icteric. Yawning is common at first. Other symptoms indicate pathological involvement, either progressive or simultaneous, of widely disseminated parts of the brain and spinal cord. Transient facial paralysis, or palsies, which may be unilateral or bilateral, are observed. Pendulous lips, irritative motor phenomena of the facial and cervical muscles may be manifested by muscular twitchings and spasms. Bulbar paralysis may predominate as evidenced by partial or complete paralysis of the pharynx, larynx, and tongue. A change of voice is noticed. Grinding of the teeth is common. The presence of decomposed food which is retained in the mouth causes a foul breath. The eyes are often half closed and ocular disturbances are present. The pupil does not respond to light normally. Walking into objects is common, which may be due either to faulty vision or to the inability of the animal to coördinate its movements.

Disturbance of the motor centers is noticed by muscular trembling, uncertainty of motion, incoördination of gait, knuckling, and swaying from side to side. Ataxia-like symptoms may be manifested. Some animals stand with the legs crossed; others assume an abnormal stance, particularly a saw-horse attitude of the legs being spread apart to insure a wider base, leaning backward or sidewise on objects for support. Animals resting in this position usually stand with the head lowered, and are indifferent to surroundings. However, they may be aroused quickly from their state of lethargy by a sudden slap (hyperesthesia) or very sharp noises. Hyperesthesia is particularly noticeable upon introduction of a hypodermic needle. This may be followed by considerable excitement.



—*After Cox, Philip, Marsh and Kilpatrick, 1938.*
Encephalomyelitis, cross-legged symptoms.

One striking symptom, always present, is rigidity of the abdominal muscles, producing a ridge extending obliquely upward from and posterior to the costal arch. This symptom is frequently referred to as the splanchnic ridge. There is apparently no abdominal pain to account for this sign. It may be due to the position of the legs and insecurity of balance.

In some cases there is complete loss of appetite. In others the appetite is retained but is accompanied by slow mastication and difficulty in swallowing. In attempting to drink, many animals frequently submerge the nostrils beneath the water level, as they

are unable to perform the act of sucking. Inconstant degrees of paralysis of the intestinal tract and bladder are due to associated myelitis. A rapid loss of weight and excessive dehydration is pronounced in all cases. Some animals appear to lose 100 pounds over night.

When the somnolent cases are aroused to forced movement, they may fall on their knees and head, to regain the standing position again by a clumsy, staggering movement. Some animals cannot be driven forward, but may move backward, finally falling over in a somersault fashion. Other animals wander aimlessly about with a tendency to walk in circles and only in one direction, either to the right or to the left, and can not be induced to turn in the opposite direction.

I have attempted to discuss the symptoms in the average well developed case. However, the greatest of variations are observed. Some animals seem to develop into hyperacute cases with complete paraplegia in four to six hours, and die soon afterwards. In this condition, some perform walking movements; others remain motionless, yet, if turned over, will not maintain that position, but will immediately find enough power to roll back, resuming the original recumbent attitude.

Some animals that are down and appear unable to rise, if rolled over, are capable of getting up, and they become favorable cases if a standing position can be maintained.

Cases terminating fatally may show a high temperature, weak, irregular pulse, and rapid respiration shortly before death. This in all probability is due to the action of secondary invaders from an overwhelmed resistance. On the other hand, some animals have a very mild case exemplified by slight dullness, such as dragging of the feet, but are apparently normal again within a few hours. Undoubtedly many animals pass through the disease unrecognized.

Stallions, in particular, and nervous animals seem to develop the disease more acutely and the percentage of fatalities is higher than in o'her animals.

Although the manifestations may be acute in suckling colts and young animals, they respond better to treatment and the mortality is not as great, by far, as in older animals. It has been reported that the mule is not as susceptible as the horse. We are not in a position to verify this opinion, but we have observed a number of cases in the mule. There is no discrimination in regard to age, sex, or breed. The maintenance of a high standard of bodily health does not necessarily produce resistance to infection.

TREATMENT

In infectious equine encephalomyelitis, there is no specific medication; the treatment is essentially symptomatic. All that may be said is that many therapeutic agents used in the treatment of the disease are empiric and have no scientific justification.

Absolute rest and quiet is imperative. This is accomplished by removing the sick animal or animals from external excitatory influences and confining them in a cool, dark, well ventilated stall. A potato cellar is an ideal place. The animal should be so confined as to allow it to lean against a wall for support. If it leans backward, a pole propped under the buttocks, to serve as a seat, should be arranged. For further support, inexpensive stocks may be built in the form of a crate. In the writer's experience, slings are contraindicated. An animal drapes itself over a sling like a wet rag and death is hastened by respiratory and cardiac failure.

Much has been said, pro and con, in regard to the use of serum. It is not logical to assume that serum will have any value after the virus, which is neurotropic, has become fixed. Therefore, it has doubtful value in the majority of well defined clinical cases.

We believe that the use of a liberal dose of serum is justifiable only in the initial stages of properly selected cases. When the temperature starts to decline, the administration of serum seems to be worthless. The usual dosage recommended is far too small. The best results, provided the value of the animal warrants it, are obtained from large doses. Administration of large amounts makes it prohibitive except in valuable animals. There are several disadvantages to the use of serum. When approximately 75

cc. has been administered, a large percentage of horses exhibit a marked systemic reaction simulating anaphylaxis. It is accompanied by excitement, cynosis, and increased respiration, and occasionally the animal falls. This is not only harmful to the horse but sometimes produces death. Heating of serum does not overcome this fault. Everything considered, the results obtained have not been gratifying in the majority of cases.

The marked dehydration that accompanies the disease can be corrected in several ways. Many horses can not drink from a bucket or trough at ground level. However, if a pail of water is elevated to the head, they may drink slowly. If this is not successful, then the administration of fluids, intravenously, into the stomach, or by enteroclysis should be resorted to. Some prefer to pass the stomach tube and inject several gallons of water into the stomach. Others inject water into the rectum. Both methods are good. Good results are obtained with little excitement by intravenous injection of two quarts to a gallon of normal saline or Ringer's solution. The last two quarts should be given rather slowly so as not to tax the heart. If the animal is not eating, 150 grams of glucose may be included in the saline solution. Owners should be instructed to give enemas every three or four hours. Overcoming the dehydration is one of the most important phases of treatment and can not be neglected. Colts, unable to nurse, may be given corn syrup dissolved in warm water or milk.

Laxatives are indicated but drastic purgatives are contraindicated. Practically all agents that will cause purgation will cause dehydration; therefore they should be avoided as much as possible. Epsom salt or any of the saline purgatives used in concentrated solutions will cause dehydration. If well diluted and given in large quantities of water, however, an osmotic equilibrium is established and little or no dehydration will result. Promotion of elimination is necessary.

As in all cases of disseminated encephalitides, harm may be done by the use of nerve stimulants, and this disease is no exception. Nux vomica, strychnine and alcoholic stimulants are to be avoided.

Bleeding and other traditional empiric treatments simply lower the resistance of the individual. It is illogical to use anti-hog cholera serum, influenza serum, bacterins or other biologics foreign to the disease. Since we have a disease that is caused by a specific virus, a specific antisera should therefore be used, granting that it has limitations. The use of the balling gun and dose syringe in medication is dangerous and to be condemned. Too often, mechanical pneumonia is the result. Overtreating is to be avoided. Treatment is worthless in so-called "downers" or animals that are prostrate.

Success is attributed to various forms of therapy when the result merely represents coincidence, with spontaneous or uneventful recoveries that the natural resistance of the individual has accomplished, in spite of treatment. Many cases will make an uneventful recovery if nothing is done. Excessive enthusiasm suspiciously betokens a passing therapeutic fad. Good nursing is ultimately of more benefit than medicants. Everything should be done to aid and support natural processes in preserving the resistance of the animal, and nothing should be done to combat it.

A great many horses, that have recovered from a typical form of the disease, may show evidence of it for months or even as long as a year. They are more or less languid, sluggish at work, and have lost their usual energy. Pregnant mares may abort during the course of the disease, or after apparent recovery.

Inexplainable relapses have been observed. One animal that had a well developed case of infectious equine encephalomyelitis three months later developed an unusual condition of being unable to drink; yet prehension, mastication and deglutition remained normal. This particular animal had to be given water daily for three weeks, by means of the stomach tube, before it regained the ability to suck.

Another horse, two months after apparent recovery, developed a peculiar condition, in that he was unable to pass through a stable door or narrow pathway without in-

clining the head sidewise, accompanied by swaying of the body, with incoördination of gait. Tractable in every way, he finally developed an extreme phobia and could hardly be driven through a door. It required about a year for these symptoms to subside.

The question to be pondered is whether the so-called "post encephalomyelitis disease" is a relapse of the virus diseases, or whether it has any relationship to it. Field and clinical observations would tend to connect them, although experimental work has failed to verify such conclusions.

PREVENTION

The veterinarian should be concerned much more with the effective prevention of this disease than its treatment. The ability of the veterinarian to control infectious equine encephalomyelitis depends upon a program of educating the live stock owner as to the true facts regarding the cause, transmission, and prevention. We gain nothing by keeping the horse breeders and owners in ignorance of the facts pertaining to any and all phases of this malady. On the contrary, we leave open a fertile field for the empirics, nostrum venders and unscrupulous commercial houses that have no outlet through the veterinary profession for their propaganda. Furthermore, serious bars to intelligent information and action have been placed in the road by the unnatural stupidity of a small number of professional men who refuse to accept the truth and transmit misleading information to their clients.

Various plans have been proposed for the prevention of infectious equine encephalomyelitis, such as screens, etc. Most of them are impractical under average farm conditions.

It is too early to predict, but so far, the practical value of protective inoculation with chick-embryo vaccine has been definitely proven to date. To vaccinate ten horses to protect one or two is probably not sound economy. Nevertheless, the economic loss in many communities in the horse industry, from death and loss of service, would pay for vaccination of the entire horse population for years to come.

During the last two weeks of May, 1938, 267 head of horses were vaccinated with chick-embryo vaccine. In spite of the fact that we had the most virulent epizoötic of the disease during the summer of that year, not a horse so vaccinated contracted the disease. In three instances, in which half of the horses were vaccinated on different farms, the disease appeared in the non-vaccinated animals. All horses on the college grounds were vaccinated, which included those belonging to the farm department and those of the R. O. T. C. artillery unit. A riding pony, belonging to the farm manager, unintentionally escaped vaccination, and was the only animal to succumb to the disease.

Lt. Colonel E. L. Nye of the Veterinary Corps, U. S. Army, reports that during army maneuvers at Pole Mountain, Wyo., the past summer, after four cavalry horses out of 486 in his charge had succumbed to encephalomyelitis, the entire cavalry unit was immediately vaccinated with chick-embryo vaccine. A march of 365 miles to Fort Meade, S. Dak., was started the day following the initial vaccination. This march required two weeks and these horses passed through numerous areas in which an epizoötic of encephalomyelitis was at its height. Not a horse contracted the disease after the first inoculation. However, he did report that the first day's march covered a distance of 22 miles and that 25 animals showed an acute systemic reaction. They had a high temperature of approximately 105°. Furthermore, the rest of the horses were sluggish and lacked their usual energy. This reaction was in evidence for approximately 72 hours, although no local reactions were observed.

Farm horses may be worked immediately following vaccination with no bad results or apparent reaction. At least, farmers and horse owners have failed to report otherwise.

The use of chick-embryo vaccine is properly intended and recommended for preseasonal use. However, its use need not be limited to this, as it appears to be a valuable protective agent during an epizoötic of this

disease. It apparently sets up a quick immunity, and normal horses can be immunized safely and effectively. In the course of the disease, 1,021 horses were vaccinated, with the result that not a single horse contracted the disease nine days after the first injection. (After the initial injection, a small percentage that have normal temperatures when vaccinated will develop the disease. Most of them will show clinical symptoms between the third and seventh days; one horse as late as the ninth day.) Most horses were mild cases. Only one showed acute symptoms and none was fatal. This has been the experience of other practitioners in this state who have used chick-embryo vaccine.

Certain precautions in the use of this vaccine should be adhered to during the course of an epizoötic. All animals should be carefully temperatured and vaccinated only if the temperature is normal. Accurate temperatures can be relied upon only after rest, preferably in the morning. Work, excitement and hot weather will cause a rise of 1 to 2°, which might be misleading Any animal showing a temperature should be closely examined and serum administered, vaccination being postponed for two weeks. Two intramuscular inoculations are made, preferably ten days apart, on the opposite sides of the neck.

In view of the seriousness of the disease, the heavy losses, largely symptomatic, the curative measures and the impossibility of eradication of the vectors that transmit it, it appears that the proper approach in the control of this disease is prevention by vaccination with a proven vaccine. As already indicated, it is too early to prophesy what the merits of chick-embryo vaccine will be over a period of years. Whether or not it will stand the test repeatedly in general, severe epizoötics, only time will tell.

SUMMARY

Colorado has experienced, apparently, two historical outbreaks of infectious equine encephalomyelitis previous to the discovery of the causative agent. The state has ecaped the disease only one year since 1932. It reappears in the same areas year after year without conferring an immunity.

The diagnosis of equine encephalomyelitis depends upon its seasonal occurrence and epizoötiology. The disease is manifested generally as a lethargic type of encephalitis, although in some instances it is ushered in by signs of increased nervous irritability. Symptoms are variable. The mortality in young animals is far less than in middle aged and old horses.

Treatment is largely symptomatic. The use of serum as a therapeutic agent is limited. Rest and overcoming dehydration and elimination are important therapeutic measures. Treatment of prostrate animals is usually worthless. Relapses are observed following the disease.

A great deal of misleading information regarding this disease has been disseminated to horse breeders and farmers throughout the country. Education of the horse owners by the veterinary profession regarding the true facts of the disease is the only method to overcome this malicious propaganda.

Prevention with the use of chick-embryo vaccine has proven to be highly effective as an immunizing agent, not only as a preseasonal vaccination, but also during an epizoötic.

References

[1]Meyer, K. F., Haring, C. M., and Howitt, B.: Newer knowledge of the neurotropic virus infections of the horse. Jour. A. V. M. A., lxxix (1931), n. s. 32 (3), pp. 376-389.

[2]Records, E. and Vawter, L. R.: Equine encephalomyelitis. Bul. No. 132, Univ. of Nevada (June, 1933.)

Equine Encephalomyelitis Kills Woman

An Associated Press report of March 29 announces the death of Mrs. Ruth Coe Ramsey, 28 years old, employée of Lederle Laboratories, Inc., Pearl River, N. Y., who had been engaged in injecting a virus into eggs in the manufacture of chick-embryo vaccine. The accident occurred in the handling of the western strain of the virus. This unfortunate fatality appears to leave no doubt as to the transmissibility of this equine virus to human beings.

Equine Encephalomyelitis Virus (Eastern Type) Isolated from Ring-Necked Pheasant*

By H. VAN ROEKEL, D. V. M., and MIRIAM K. CLARKE
Massachusetts Agricultural Experiment Station, Amherst, Mass.

TWO RING-NECKED pheasants were received by Dr. F. R. Beaudette of the New Jersey Agricultural Experiment Station, New Brunswick, N. J., on September 16, 1938. These pheasants were submitted by a game breeder located in New Monmouth, Monmouth County, N. J. Losses in the pheasant flock resulted from a disturbance which was suspected might be the disease designated as infectious avian encephalomyelitis (epidemic tremor). Since this disease was under investigation at the Massachusetts Agricultural Experiment Station, brain material was submitted to the Department of Veterinary Science for identification of the infective agent.

A saline suspension of the pheasant brain was inoculated into six five-day-old Rhode Island Red chicks. The third day after the inoculation, three chicks appeared sick. Two of these died on the fourth day and the third was killed for inoculating material. Two chicks appeared sick on the fourth day and died two days later. The sixth chick survived the effects of the inoculum. The clinical manifestations of the disease in the experimental chicks immediately suggested a type of infective agent which produced a clinical picture differing from that caused by infectious avian encephalomyelitis strains that were investigated. Subsequent serial intracerebral inoculations into chicks employing brain-saline suspensions revealed that the infective agent caused visible signs of disturbance within twelve hours after injection. Death followed within 18 to 36 hours after exposure. One hundred per cent mortality was observed among one- or two-day-old inoculated chicks, while in some instances in one- to two-week-old chicks no visible evidence of the disease was noted. The infective agent has been passed through ten series of chicks with no apparent change in the clinical manifestations produced. In one instance, the infective agent has remained viable in a brain-saline suspension held at 8° C. for a period of 71 days without an apparent loss in virulence. In a second instance, the infective agent failed to produce the disease after being stored for 71 days. On another occasion, three brain-saline suspensions of different ages (94, 75 and 52 days, respectively) were inoculated intracerebrally into two-day-old chicks. Among the chicks in the three groups, the majority manifested symptoms within 24 hours after inoculation and all chicks except one succumbed within 48 to 96 hours after exposure. The one surviving chick inoculated with the 94-day-old suspension was killed on the sixth day. Its brain was tested for the presence of virus yielding only questionable results.

RING-NECKED PHEASANT INOCULATION

One pheasant given intracerebral and intranasal inoculations with original material failed to exhibit evidence of disease. A second pheasant was inoculated intracerebrally with a brain-saline suspension prepared from a second serial-passage chick. In this instance, positive transmission of the disease was obtained. Within 24 hours after the inoculation, the bird manifested droopy wings, a reluctance to fly and an ataxia. A second intracerebral inoculation was given 48 hours after the first exposure. Two days later the bird was prostrate and comatose. Inoculum prepared from the brain of this pheasant was injected intracerebrally into a third pheasant, which developed symptoms similar to those observed in the second bird. Death followed six days after the single inoculation.

*Contribution No. 344 of the Massachusetts Agricultural Experiment Station.

ENGLISH SPARROW INOCULATION

Six English sparrows (*Passer domesticus*) were inoculated intracerebrally with brain-saline suspension prepared from a ninth serial-passage chick. The sparrows manifested symptoms within 16 hours after injection. The birds appeared at first less active, which was followed by somnolence, inability to fly, marked weakness, prostration, coma and death. Death occurred within 19 to 24 hours after the inoculation. In this trial, the course of the disease appeared very similar to that observed in inoculated day-old chicks.

In a second trial, five sparrows were inoculated intracerebrally with a brain-saline suspension which had been retained at 8° C. for 30 days. Only one sparrow manifested marked and definite symptoms of the disease within 24 hours. Its brain was used for inoculum and inoculated into three sparrows and four chicks, all of which succumbed within 24 to 48 hours. The four remaining sparrows which exhibited no symptoms 48 hours after the first inoculation were given a second injection. All but one sparrow manifested symptoms and succumbed. However, death occurred later than in previously inoculated sparrows, which was in a large measure attributed to the loss in infectivity and virulence of the agent due to storage. The one sparrow which did not succumb was killed. A brain-saline suspension prepared from this sparrow revealed the presence of the infective agent which was lethal to day-old chicks. Sparrows may be regarded as highly susceptible to this infective agent. Ten English sparrows, inoculated intracerebrally with a brain-saline suspension prepared from a chick affected with infectious avian encephalomyelitis (epidemic tremor), exhibited no clinical manifestations of that disease.

GUINEA PIG INOCULATION

Brain-saline suspension inoculated intracerebrally into guinea pigs produced definite clinical manifestations which terminated with death three to five days after exposure. A striking feature observed in inoculated guinea pigs was the sudden febrile reaction. Within 18 to 24 hours after inoculation, the temperature mounted several degrees. The elevated temperature persisted for 24 to 30 hours and then declined to below normal. Further manifestations were inactiveness, progressive weakness and paralysis, intermittent muscular tremors of the extremities, loss in weight, and coma followed by death. One guinea pig inoculated intraperitoneally manifested on the first day after inoculation only an elevated temperature, which subsided to normal on the third day.

CROSS-IMMUNITY TESTS

The guinea pigs employed for cross-immunity tests were in part supplied by the Lederle Laboratories, Inc., and the Department of Bacteriology and Immunology, Harvard Medical School, to whom we express our sincere appreciation.

Fourteen guinea pigs (six immunized with the Eastern type of equine encephalomyelitis vaccine, four with the Western type vaccine and four normal controls) were injected intracerebrally with the infective agent. All the controls and all the guinea pigs immunized against the Western type of virus died within three to six days after inoculation. All but two of the guinea pigs immunized against the Eastern type virus survived the inoculation. However, all the survivors manifested a temperature rise within 24 hours after the injection similar to that observed in the normal controls and those immunized against the Western type virus. The two pigs that succumbed and that had been immunized against Eastern type virus expressed some degree of resistance to the inoculated infective agent in that death did not occur as soon.

NEUTRALIZATION TESTS

Through the kind coöperation of Dr. LeRoy D. Fothergill, Harvard Medical School, to whom we are indebted, we were able to have the infective agent subjected to neutralization tests employing a human serum obtained from an individual who had recovered from encephalitis of the Eastern equine encephalomyelitis type. It was observed that the virus was neutralized by

human serum and also completely neutralized by a hyperimmune horse serum of the Eastern type. Hyperimmune horse serum of the Western type exhibited very slight neutralization. Dr. Fothergill also observed that when the infective agent was inoculated into mice, symptoms similar to those produced by the equine encephalomyelitis virus were manifested.

These results definitely reveal that the infective agent recovered from the pheasant brain appears identical with the virus that produces the Eastern type of equine encephalomyelitis.

It is of great significance to note that the virus of equine encephalomyelitis has been observed and identified in several new hosts in nature during the past year. The distribution and intensity of this infection among birds has not been determined with any degree of completeness, but it is of interest to note that the natural cases[1, 2] positively identified, including the one reported here, occurred at about the same time in three different states (Massachusetts, Connecticut, New Jersey) along the Atlantic seaboard. Other suspicious outbreaks among pheasants were reported in Massachusetts and Rhode Island at the time the disease manifested itself in man and horses in this area.

Since the natural prevalence of the disease has been identified in several species of hosts, intensive search should be made for the presence of this infection among other birds and mammals as well as insect vectors which play a possible role in the dissemination of the virus. Latent infections in susceptible hosts [3, 4, 5] and possible carriers among apparently nonsusceptible species command investigation for effective control and prevention of the disease.

SUMMARY

The occurrence of equine encephalomyelitis virus (Eastern type) among pheasants in New Jersey has been observed and identified. The English sparrow (*Passer domesticus*) was found highly susceptible to this infective agent through experimental inoculation, and should be added to the list of hosts which may contract the disease.

Prenatal Hydrocephalus

Although pathological rather than teratological, these prenatal cases of hydrocephalus are generally named monstrosities in clinical work. The one illustrated herewith occurred in the practice of L. H. Brown, of Elkhorn, Neb., who describes the fetal carcass as normal in every other respect. The presentation was anterior and

—Courtesy Corn States Serum Co.

Prenatal hydrocephalus.

the position normal. Owing, however, to the enormous size and unyielding nature of the enlargement, the puerperal damage inflicted in delivery was fatal to the dam. Bone comprised a too large percentage of the total volume to reduce by evacuation of the liquid and crushing of the walls.

(*Continued from preceding column.*)

References

[1]Tyzzer, E. E., Sellards, A. W., and Bengtson, B. L.: Sci., lxxxviii (1938), p. 505.

[2]Fothergill, L. D., and Dingle, J. H.: Sci., lxxxviii (1938), p. 549.

[3]Giltner, L. T., and Shahan, M. S.: Sci., lxxviii (1933), p. 63.

[4]Giltner, L. T., and Shahan, M. S.: Jour. A. V. M. A., lxxxviii (1936), n. s. 41 (3), p. 363.

[5]Remlinger, P., and Bailly, J.: Compt. Rend. Soc. Biol., cxx (1935), p. 1067; *ibid.*, cxxi (1936), p. 146; *ibid.*, cxxii (1936), p. 518; *ibid.*, cxxiii (1936), p. 562.

Equine Breeding Hygiene*

By W. W. DIMOCK, D.V.S.
University of Kentucky, Lexington, Ky.

DR. GILYARD, LADIES AND GENTLEMEN: It has been difficult for me to choose a suitable topic for my address. Since this is the Section on General Practice, I believe it will be quite all right if I talk to you on the principals of equine breeding hygiene.

I have a feeling that there is a great deal of valuable and very definite information about the control and prevention of breeding problems in mares that is not being used. As essential as research, new ideas and new facts are, we must not let the application of those definite facts and principles lag.

I realize, too, that the horse breeding during the last 20 or 25 years has been on the decline, but it has certainly improved somewhat during the past four, five, or six years, and there is an increased interest in the production of horses. They are shipping to Kentucky many carloads of mares from the West, to try to get back into the mule business which Missouri took away from them many years ago.

It would really take about 2½ hours to tell this story properly, but as a basis of what I am going to tell you about the application of some of these principles of breeding hygiene, I do want to give a few figures on the results of the examination of brood mares and of fetuses, that is, in the case of abortion, navel ill, joint ill, and pyosepticemia—infections of the newborn.

We have a record of about 4,000 mares—mares that were not in foal, mares that were examined to try to find out why they did not get in foal the past breeding season. Of these 4,000 mares, we find that about 65 or 66 per cent apparently had a normal genital tract. But we find in 25 per cent—that is, one out of every four—that these mares that were examined had streptococcic metritis. We will take that 66 and that 25, and that leaves a small percentage of other sorts of miscellaneous diseases up to July a year ago.

During the past year we examined between 250 and 300, and there were 62 per cent that had no infection, and 28 per cent had streptococcic metritis.

In regard to the fetuses that come to us for examination—and we are only reporting on those that had a detailed bacteriological examination—we feel the diagnosis was correct. There were 43 per cent that did not have anything. But there were 20 per cent of the cases of abortion that came to us that had the abortion occur as a result of streptococcic metritis. About 24 per cent of these cases of abortion were the result of what we call virus, or epizoötic abortion. We are quite convinced that there is a filtrable virus disease of mares that results in abortion. We have transmitted this by direct inoculation and by a filtrate. This spring we had a number of these foals in the laboratory, and as we were rebuilding our postmortem room we held these autopsies on these fetuses in the operating room which is next to my stable. I had a saddle mare in there, and the same boy who helped with these fetuses takes care of the mare. One morning we found she had aborted, aborted from virus abortion, as we call it. Thus, there were 43 per cent negative, 23 or 24 per cent virus abortion, and 20 per cent streptococcic infection. Last year there were 19 per cent streptococcic infection, 23 per cent negative, and 14 per cent virus abortion.

There were 500 foals that had carried full term. Twenty-five per cent of the foals died of streptococcic infection, navel ill, or joint ill—25 per cent within 30 days. Thirty-five or 36 per cent had what is spoken of as *Bacterium viscosum equi* infection, that is, up to July, a year ago.

*Extemporaneous lecture delivered before the seventy-fifth annual meeting of the A.V.M.A., New York City, July 5-9, 1938.

During the year, 20 per cent of these foals had streptococcic infection, 48 per cent were negative, and 20 per cent *B. viscosum equi*.

I am particularly anxious that you should keep in mind the streptococcic cases. Twenty-five per cent of brood mares are suffering with extreme metritis. Twenty to 25 per cent of abortions are due to streptococcic infection, as are also 20 to 25 per cent of navel ill. If there is any one infection that infects brood mares and foals and fetuses, in foals that you can control by hygiene, it is this streptococcic infection. If you want to increase the percentage of pregnancies, if you want to increase the chance of having a live, healthy foal at term, the mares admitted to service must be examined, and that means that the practitioner has to know how to examine the mare—speaking strictly of genital infections, he has to tell the difference between a normal mare and an infected mare. You can learn to tell very readily the difference between a perfectly normal, healthy mare, and a badly diseased one. But there are those borderline cases that you can not always detect from a clinical examination, and you have to resort to some method to confirm your opinion. Some mares have no bacteria present, no infection on examination, and yet they look a little red and congested, showing symptoms of infection. In those cases, we always resort to making cultures from the cervix. We do that by using a vaginal speculum and a platinum needle with a long handle, pass it over a spirit lamp and into the cervix. You will be surprised at how accurate that will be. Many of them will be negative, and from the infected you will recover the organism from the cervix. This has been of great benefit to the breeders of Kentucky and adjacent regions.

If I were going to say anything to these younger men who may later get mixed up with the horse-breeding problems, I would say the first thing to do is to learn to tell the difference between a normal, healthy mare, a mare that has metritis, and those that have other diseases. Then I would devise some way to find out something about the borderline cases, the cases on clinical examination, where you are unable to make up your mind whether that is a redness, an inflammation, and congestion due to infection, or something that does not amount to anything. That is the basic thing to know about the mare.

Now, of course, genital infection is not the only reason that the mare fails to get in foal. There are ovarian troubles and an unbalanced endocrine system, and possibly many other things. I think the problem of nutrition comes in. I was interested in what they had to say last night and other times here about nutrition in animals. Of course, we are a little bit conceited in Kentucky about nutrition. We feel that probably our animals do not suffer from any serious cases of malnutrition. The mares are out on the pasture pretty nearly 12 months in the year. We have pretty good pasture for nearly 10 months in the year, and in addition to the grass that we grow on our own pastures, we ship in hay from Wisconsin and Michigan and Ohio and Indiana, and of course, grain, and you would not say there was anything the matter with that hay and grain. So, considering our pastures and your hay and grain, I would say that perhaps our nutrition is quite all right. I do think you can go this country over and you will find that there is an opportunity to improve the ration of horses by balancing it with a greater variety. I think there is an opportunity to do that all over the country.

There have been one or two places—I am not going to mention names; I am just giving instances; I can furnish the evidence If I am put on the witness stand—where they have taken the procedures that we have worked out at the University, and gone into these breeding establishments and increased the percentage of pregnancies anywhere from 10 to 20 per cent, and they not only increased the percentage of pregnancies among the mares maintained, but they carried through and produced normal foals. There was one place that had 172 mares pregnant. There were

172 mares foaled, and they weaned 172 foals. That is pretty good breeding efficiency, and they did it simply by the application of the principles of equine breeding hygiene.

The streptococcic infection that causes metritis, abortion, and navel ill is exactly the same streptococcus, and it always enters the mare through the genital tract. Therefore, it is either in the mare when she is bred, or it is introduced at the time of service, and we have controlled that. Now, remember, you are controlling a condition that causes one-fourth of the diseases due to streptococcic infection. Twenty to 25 per cent of your cases of abortion are due to streptococcic infection, and 20 to 25 per cent of your navel ills are due to streptococcic infection. These are the percentages. You can control that, and you can raise your live foals 10 per cent—come at term and go on and grow—just simply by being clean.

You can use ovarian extract and estral hormone and gonads and those things, but they will defeat you unless you can determine the kind of case you can use them on. Why do you want to give those things to a mare that has streptococcic infection or metritis? I think you would get the same results by passing a magic wand over them.

We have practiced examining every mare before she is bred, and no mare, unless she is perfectly normal and healthy in the genital tract is admitted to service. Make a practice of having mares properly prepared for service. The tail is bandaged and she is washed up and cleaned. The stallions are properly taken care of. They are given a bath before and after they serve the mare.

They have taken this procedure, and they have absolutely made it work. Many fellows will say, "Well, this horse will not be bossed, and he is a lot of trouble, and he is vicious," and all that. I can take you to a breeding establishment in Kentucky where they have 8, 10, or 12 stallions, and 200 or 300 mares on the farm, besides hundreds of others that are brought there for service. They go through a great deal of what might seem unnecessary procedure to keep things clean. But when these stallions come into the breeding shed, the mare

Where Your Eyes Will Be Focused August 28 to September 1, 1939

MEMPHIS ON THE MISSISSIPPI

The many buildings towering into the sky tell their own story of the progress and achievement of this famed city of the South.

is there and she is ready—hobbled, the tail bandaged, and cleaned up. When the stallion comes he gets ready, and without being touched he backs into the corner and takes his bath. Then like a gentleman he comes up and serves the mare, and when he is through, he backs into the corner and gets another bath and goes into the barn.

You can train him just as you can train a dog. They are no trouble at all. I was talking to the veterinarian in charge of this breeding establishment just last week, and he said, "This has increased the percentage of pregnancies on this farm 10 per cent."

If out of 100 mares, 80 mares are in foal, that is pretty good. Seventy is not so good. When I went down to Kentucky—(and I am not bragging)—I think the average was about 60 per cent. The average now is about 70 per cent, and on the good farms, 80 per cent. In certain groups it may be higher. I know a man who last year had three mares, and they were all in foal. Another had six, and they were all in foal.

It is worth while. What the horse breeding industry needs is to do these things I am talking about, especially looking after these general infections. In the future they are going to practice artificial insemination, and transport the semen, and perhaps that will not make much difference. But we older fellows still feel that the old way is just as good, perhaps better in some respects. Therefore, there is still an opportunity, and with all the good features and advantages that there may be in artificial insemination, and all the good that we may derive from the use of the various hormones and concentrated vitamins and things that we are going to have to administer to mares, there is still the question of knowing whether that mare is a subject that needs that vitamin and that hormone, or whatever it may be. There is nobody in the world that can tell whether the mare needs it or not but you and I. That is our business, and we should make it our business, but we should not let this thing get going in a way that is going to have a come-back.

We found some years ago that there was quite a lot of difference of opinion among owners and managers of farms and veterinarians as to whether a mare was in foal or not. The operating season is very short—from February to the 15th of June. They feel that they are going to have a race horse or a trotting horse or a saddle horse and they would like to have them come earlier, because they have to compete. If the foal comes the first of January, on the first of July he has to compete in the same group. Six months is quite a difference when they compete in the two-year-old class. They like to have these mares in foal early, but there is this: They do not know whether these mares will foal or not.

If a mare is not in foal, and you are going to do anything for her, the time to do it is from September to January. If you wait until January, you go out like the Colonel does in Kentucky. He has a couple of mint juleps and looks the old mare over, and says, "She's a little more filled out than she was yesterday." They squint and poke her in the belly with a cane, and one fellow says, "Well, George, I will bet you $100.00 she is in foal." When that happened, the man I have in mind did not have the nerve to take the bet. He called me up the next day and I went up there. He said, "Doc, I have a chance to make $100.00, or I had it yesterday, and today I wish I had taken it. I think the old mare is in foal."

I stuck my hand in her rectum up to my wrist, and there was a foal 10 months old. His head was almost ready to come out. I tell this simply to illustrate how in the olden days, these old fellows could not tell at 10 months whether the mare was in foal, by looking at her.

If she was in foal, that is perfectly all right, but if she was not in foal, it is a good idea to have known last September whether she was, and that brings up the question of examining mares for pregnancy. I suppose that 90 per cent of the men in this country make a daily practice of examining cattle for pregnancy, but there are still a lot of people who own brood mares in this country who are scared to death. Even up here a few years ago at

the International Veterinary Congress, they brought a man over from Europe who said, "The rectal examination is perfectly all right, it is harmless, but for heaven's sake, do not put any speculum into the vagina of pregnant mares. It will cause them to abort."

I have put the speculum in the vaginas of hundreds of mares in foal. It did not cause them to abort. I have had men stand on the street corner, 15 or 20 years ago, and they would say, "I understand you say that you ought to put your hand up a mare and examine it for pregnancy." I said, "Certainly. I believe it is the right thing to know in September." One man said, "You can not do it for me." The next day he called me up and wanted to know how soon I could examine one or two. It has gone from that stage until today they serve them yesterday, and they want to know today whether it is in foal.

It is true that I have worked largely with breeding establishments where they have anywhere from ten to 200 mares. Most of them are owned by men here in New York, Philadelphia, Detroit and Chicago and other places, who have plenty of money, and they come down to Lexington, buy a big acreage and go into the breeding of horses, but those men have been very generous in making their breeding establishments available for use by us in making observations. If you ever get a good, keen business man and explain this proposition to him, and he gets sold on it, he is as keen as anybody, but it does not make any difference to him if his mares do not get in foal. He will have bacon and eggs for breakfast just the same.

It is the poor devil who is on the farm that we have to help. He has been to the bank and borrowed $200, $300 or $400 to buy a pair of mares. The idea is that he

can raise a couple of foals, perhaps, and it will bring in a little money to pay the taxes, and maybe buy the wife a new pair of shoes. It means a good deal to him if his mares get in foal, or if the foals die or the mares abort. That is the fellow to protect. While we have used, in our immediate neighborhood, these large farms owned by wealthy men to run these experiments and make the examinations, the idea is to make the observations and take it out and use it for the fellow who needs it, and try to save his foal, try to get his mare in foal, and try to have her carry through full term and give birth to a healthy foal.

Then the problem is about half over. He will probably turn her out in a patch that has been used by foals for years and infested with parasites, and he will probably die. You ought to know about that, too. As well as knowing that the mare is healthy and normal, it is well to know the stallion is healthy and fertile. We have a record of some 100 to 150 stallions, and at the time we finished with the 100, 10 per cent of the studs—and this includes all kinds of studs, all breeds—were absolutely sterile.

Just by examining the semen of these stallions, examining the fresh and smear preparations, you get the history and performance of the stud, and you can classify them. We had 10 per cent that were absolute blanks. Then you can put them into four other classes—the excellent, good, fair, and just now and then. If you can examine the semen and study it, you will find that your observations, from this microscopic examination of the semen, will absolutely check with the breeding record of the stallion. So you have to consider both the mare and the stallion.

I want just to voice the idea here that I think we, as professionals, should do everything we can to tell stud growers and managers and others that have to do with horses to keep their dirty hands out of mares, so far as this practice of opening them up is concerned, and so far as practicing artificial insemination is concerned, on their own. I have examined a good many hundred mares, and I never saw any, except maybe one or two, that needed opening up. I have seen one or two mares that had to foal, and a little film had grown over the cervix. I broke this with my finger, and the next time she had gotten the foal. You would not have known this if you had put the speculum in, but there was a complete partition of fibrous tissue grown across the cervical channel, and of course nothing got in there. But there is not 1 per cent that needs opening up. They do more harm than they do good. You might introduce an infection that way.

Streptococcic infection in foals is a prenatal infection. As I said, this is either in the mare when she is bred, or it is introduced at the time of service, and the result may be that the mare does not conceive, or that she aborts, or the foal has died or is a cripple the rest of his life. So there is the one infection that perhaps represents the majority of the trouble in breeding problems in mares, that can be absolutely prevented by being clean, and by knowing what constitutes a normal and what constitutes an abnormal mare.

We have done a lot of work on abortion. I have just spoken about streptococcic abortion. We have this virus abortion, and I am sure that you have it in the central West, and I think you have it all over the United States. We get a good many reports from biological stations and other places. They will say that one fellow had 8 or 10 mares abort, and every one was negative of equine contagious abortion. You do have these incidental cases of abortion in mares that are accidental here and there, but when you have 25 or 50 or 75 per cent of your mares abort, and the fetus is absolutely bacteriologically negative, the mare cleans up just as rapidly as if she had had a normal foal; then there is some cause, and it is not accidental.

There was a breeding establishment in Lexington, and there were some 32 mares that foaled. Twenty-four of them aborted. The manager was a rather unusual fellow, peculiar in many ways. He went over to the barn one night. He had a stable that held a 16-stall barn. Every mare stood there eating her oats, with the head up in the feed box. Everything was all right.

He said, "I guess nothing will happen tonight." He was under an intense strain, and when he went into the house he said, "I will just relax a little." He had gotten a highball mixed, and his feet on the footstool, when the telephone rang.

The man said, "Mr. Smith, a mare over here just slipped." The manager said, "What barn are you talking from?" The reply was, "Barn No. 2." He said, "I just came from there. That is impossible." The man who was telephoning said, "I think she has." The manager walked over there, and found she had not taken her nose out of the feed box. She was standing right where he had left her 30 minutes before, and there was the fetus and the afterbirth, and she had cleaned up just as rapidly as a normal case of parturition.

I do not mean to say that you do not have suffering occasionally from metritis and after an abortion, but that is the characteristic thing about that. There is no preparation. There was a mare eating—she is perfectly all right. The next minute she is stripped. You can culture the mare, the afterbirth, the fetus, and the internal organs, and you will not find an organism. That is what we believe is a filtrable virus. That has been partially confirmed in some European countries, and I think perhaps in this country.

I do not know what to do for it. We have tried to use a convalescent serum—we have tried to make a serum, and in cases where we did that, they think it has done some good. It is too early to tell.

We have a good deal of trouble in knowing when the mare is in heat. We have mares that are bred early in the spring, and they do not come back in season, and they are not in foal. We occasionally have mares that are bred and conceive and they will show signs sometimes during the spring, and sometimes they are re-bred. It may result in an abortion, or it may result in a mare being premature. So this early examination for pregnancy is valuable. On most of the farms where they have 20 to 50 mares, they start to breed the first of February, and at the end of 60 days they go over every mare that has been bred 30 days. Then they wait two or three weeks, and take the next group, just like you do in cattle. By the middle of June, or early in June, you have been over this group of mares three times—not the same ones, understand. You pronounce some of them in foal and turn them out. The breeder has taken to this because he knows from experience that a lot of mares that are not in foal, nursing foal, do not come in season, and as a result the mare comes up barren the next fall. If he can find out at the end of 30 to 60 days, early in the spring, that the mare is not in foal, it is possible to do something, perhaps, to bring her in season and get her bred before the close of the breeding season.

I was out at a place a year or two ago and examined some mares of a friend of mine. We came along in the barn. Here stood an old gray mare. I stopped and he said, "No, we are not going to examine that one." I said, "What is the matter?" He said, "She doesn't belong here. That is a mare we lease, that we are paying a big price for." I said, "I should think that is the mare you would want to examine." He said, "The fellow who owns her does not believe in that kind of thing. I do not feel that I want to take the authority to have her examined."

My curiosity was aroused right away. I said, "I have examined a good many mares for you and no harm ever came of it. You back her up. The owner does not need to know about it. I do not care if you are paying him $2,000 or $5,000 a year for the lease of this mare. That is all the more reason why you should have her examined." She had been bred exactly 60 days. He backed her up to the door and I examined her, and I said, "She is not in foal." He was dumbfounded. Here it was the first of June. He had to pay the lease just the same. So we gave some of these good extracts that our biological laboratories make. She came in heat, we bred her, and she got in foal.

Suppose I had not examined her. It would not have made any difference, of

course, since the owner has plenty of money. He can afford to pay the lease, whether she had gotten in foal or not, but it is nice to know whether the mare is in foal or not.

There was a friend of mine who had a saddle horse. He bred a mare last spring, a year ago, sent the mare home, and sent word to the man, "This mare is definitely in foal." They watched her all winter, and then the owner said, "She is not in foal." Another man said that he would bet him the mare is in foal. They bet, and the next day she aborted the fetus.

All right. Let those things happen. It does not matter, because it makes an impression on that fellow. He will not do that again. He called me up last Friday and begged me to come over and examine the mare that he had bred this spring. He said he did not want that to happen again. It takes a jar to get some of these fellows straightened out, but that man is ready to go now.

I think, gentlemen, there is a wonderful opportunity to carry on with these men who are engaged in the breeding of horses. You can do them a lot of good. I know there are men in Pennsylvania interested in horse breeding. A good friend of mine from Pennsylvania came down to see me. He was telling me about a man out there that I think has 16 draft horses, mares. The year before, 8 of them were in foal. That is 50 per cent. But of all of the mares that were in foal, the foals died of navel ill. It is nothing in the world but a question of hygiene, largely, to prevent those things, and the thing that I am interested in is that we should get out and absolutely demonstrate that these things can be prevented.

You can go into any group of mares and increase the percentage of foals from 10 to 15 per cent in a year or two, and you can increase the livability of the foals and prevent these things through the simple principles of hygiene, by clinical methods. You do not have to have any expensive apparatus.

We have a good deal of trouble with ovaries. The ovaries are cystic or some of them have tumors, and it is a question of what to do. There is a friend of mine who worked with me some years ago, a veterinarian, now on his own, working with a large number of mares. He made the statement that he did not believe that tapping the ovaries is of any value. I was surprised to hear him say that, and it kind of worried me. Perhaps I was not doing the things that I should do.

The owner came in one day and told me about a mare. I examined it. He said, "Dr. So-an-so told me last spring that this mare's ovary ought to be tapped." I said, "I thought he did not believe in that kind of thing." He said, "That is what he told me." I asked the veterinarian, and he said, "Yes, I thought they ought to be tapped."

I do believe that a mare that has a cystic ovary or a fibrocytic ovary will derive a great deal of benefit from having the ovary tapped. I tapped a mare's ovary in England some years ago that was 6 or 8 inches across, and I took out eight ounces of fluid, in August. I was there again the next year and the mare was not in foal. The thing had filled up again, just as big as it was before. I said I wanted to tap the ovary again. This mare dated back on this farm 126 years. It was the last one, and he wanted to get a filly out of it. We tapped the ovary the second time, and he bred her and she had a filly.

I believe in it, and I do not say that you should abuse the privilege, because you can go through the formality of tapping the cystic ovary of the mare, and nobody knows whether you tapped it or not. I use a long needle, and I push it in, and if there is any fluid, it runs out. I use a lot of extracts. I use ovarian extract and hormones and gonads and everything else. I have used them where I can get along just as well without them, but it gets to the point sometimes where you have to do something, and for the benefit of those who believe in extracts, and for the benefit of those making extracts, I am going to tell this little incident:

A man came down to Lexington and bought a mare for a friend of his, on commission. He paid $4,500 for it. She was knocked down to him. I was sitting beside him on the bench in the sale ring, and he said, "Let's go down to the barn and take another look at this mare." I went up there. The catalogue had said that the mare was bred in June. We asked the boy when the mare was bred and he said she had not been bred. We asked him why not, and he said she had not been in heat. The buyer scratched his head and said, "That is a situation. I sit here and buy a mare for $4,500, and she not only is not in foal, she has not been bred, but she will not come in heat."

She had a belly on her, and he said, "She looks like she might be in foal." The stable boy said, "We thought sometimes she might be in foal." It ended up with my examining her. I found she was not in foal. He turned her over to me, and this is where the extracts come in.

I started to inject that mare on the 20th of May, and I used several different kinds. I remember she had not been in heat since the first of February and to the 20th of May. I started to inject her with those various products. I will not mention their names, or who made them. She came in heat on the fourth day of June, she was served on the fourth and the sixth, and she got in foal. She had twins and both of them came alive, and were shipped to England. I think that is saying a good deal for extracts. I do not believe that was an accident.

I use them, but I never use them on a case unless I have made a thorough examination. I know my mare is normal and healthy in the genital tract. I know the stallion she is being bred to is fertile and healthy, and I know the ovaries have not a diseased condition that in a general way will prevent conception. If you will select your cases to use these various products, you will get results. If you have gone around using them promiscuously, your percentage of success will be small, and you will kind of lose faith in them.

There is just one other thing, and then I am going to stop. We have found that a lot of mares—I do not know that this applies so much to the draft mares and common farm mares, but I am inclined to think that perhaps it does—even these thoroughbreds that are raced, are very sunken and relaxed in the vulva, the peritoneal region. Especially in mares that have had a large number of foals, the lips of the vulva are all stretched out, and there is apt to be a little seepage into the vagina, and you have a chronic vaginitis. If you find mares of that kind, you make your rectal examination and find the uterus is in fairly good shape, it is well contracted, but there is a little evidence of vaginitis. Maybe you culture it and get some bacteria from the cervix.

It is a trick to take the vulva, peel off the mucous membrane—you can come down about an inch or an inch and a half between the skin and the mucous membrane and cut it off. You take two sutures—and be sure your lower suture goes through the mucous membrane—put on some metal clips, and let the mare go. If you do that, in the fall, to these mares that are a little open and have a little vaginitis, put these sutures in and leave her alone, you will find that by next spring she is clean and all right and she breeds and probably gets a high percentage of foal.

There are many other things that I wish I had time to talk to you about, but it is getting late in the afternoon. There are a number of men on this program, and I simply want to thank you for your kindness and patience.

Discussion

DR. SILAS KEMPF (Roanoke, Illinois): I want to ask Dr. Dimock whether or not he thinks that feed may have a lot to do with abortions. I am speaking of hydrocyanic acid, in feed—or as we call it in Illinois, forage poisoning.

DR. DIMOCK: So far as our experience goes, I could not say that we have any number of these cases of abortion that can be traced to the feed. We have some abortions that it is not possible to analyze and put your finger on a definite, specific cause, but those are usually just one mare here and there in a group of 10 or 20 or 30, and if no more cases occur, you generally forget about them. If it were due to anything in the feed, in the way of a chemical or toxin, it seems to me you would have more than one case in the group.

We had a mare this spring that aborted. It was shipped in from Oklahoma. She got sick some weeks ago, and the question was whether she had encephalomyelitis or botulism or intoxication, or what. She was sick for a month or six weeks. She started to get better, then had a relapse and died. Just before she died, she aborted. You could not say she aborted from the sickness, the toxin in the beginning, because she did not abort until almost the night before she died, and I think that is more or less of an accident.

I could not say. We have any number of cases of abortion that would be due to toxins or chemical products in the field.

DR. FERGUSON: Do you have much endometritis—do you find much in mares, Doctor—and if you do, how do you handle it?

DR. DIMOCK: You have reminded me of something that I intended to take up. We do have, yes. I should have stated that the origin of a lot of our streptococcic metritis is in the foaling mare. Now the normal, healthy barren mare is rather resistant to streptococcic infection. I mean you can do a lot, you can put your hand in there and serve her without regard to cleanliness, and she is pretty resistant. But your foaling mare, 50 per cent of them have strep around the cervix from the first to the ninth day following parturition. A certain per cent of them, if you leave them alone, will clean up, but they do have a little endometritis. We treat those mares this way:

Most breeders make a practice of examining all their mares that foal before they are admitted to service. They examine the barren mare and the foaling mare. After you have had some experience, about the fifth or the seventh day you can put a speculum in a foaling mare and you can judge pretty well just from the visual examination whether or not she is going to be all right on the ninth day. But those that do show evidence of infection, we douche liberally. Then I use that same old thing that I have talked to so many of you for the last 20 years about—I use the mineral oil with a little iodine. I have put that up into a mare up until the seventh day. We douche the mare and drain her as completely as it is possible to drain, even using a little suction with your pump, if you have to.

I started out by putting in 8 ounces, but now I put in 16 ounces of high-grade mineral oil, with just enough iodine to give it a red wine color. I put a pint in there and just leave it in there. It is soothing, and it is something that bacteria do not grow in. The mare will throw it out. I do not recommend that you put in oil in a mare on the seventh day and breed her on the ninth day, because you will not get by with it in many cases. But I have done it. I have put it into the mare's uterus the seventh day after she foaled and bred her on the ninth day, and she conceived and produced a normal foal. But that is pretty close work.

We have an understanding that these mares that foal in January and February and March should be treated strictly. If there is any evidence of endometritis, if they are not making a normal recovery following parturition, we always hold that mare over. Let her skip that nine-day period, and when she comes back in season, time is all she needs.

But when you get up to late in May, and the breeding season for this particular group is going to close on June 15, you cannot very well hold the mare over, so we are very strict early in the season, and we always give the mare the benefit of the doubt late in the season. You can douche them and oil them any time from the time they foal, up to the seventh day. If it were in May, I would not breed her on the ninth day. I would hold her over. If it is the first of June, give her a chance.

Colin's Summer Sore Paste

In addition to protecting summer sores against flies and physical injury from rubbing and biting, a protective paste described by Captain Colin of the French army[*] is said to possess more than usual curative properties. The paste is composed of creosote, 5 grams; salicylic acid or salol, 20 grams; oxide of zinc, 20 grams; plaster of Paris, 100 grams; water, 150 grams.

The plaster is mixed with the water in a porcelain mortar and the other ingredients stirred in in the order named. After cleaning the wound with alcohol and ether, tincture of iodine or copper sulfate solution, the paste is plastered over it with a spatula and covered with a thin layer of absorbent cotton. A second layer of the paste and another layer of cotton may be added to reinforce the coating. If the paste hardens too soon in the process of applying, a little more water is added.

Cures in a few days from this simple treatment are reported even under unfavorable conditions: extremely hot weather and numerous aggressive flies.

The formula is recommended also for the protection and healing of other surface wounds.

The author points out (sagaciously), as one would suspect, that the plaster may detach if not properly applied or subjected to injury, but inasmuch as summer sores are a veritable terror in veterinary practice another remedy is always welcome.

[*]Bulletin de l'Académie Vétérinaire, xi (Apr. 1938), pp. 182-183.

In the state of Alabama, it is unlawful to trade mules after dark.

Calfhood Vaccination Against Bang's Disease: I. Effect on Agglutinin Titres and Results of First Pregnancies*

By JOHN G. HARDENBERGH, V. M. D.

Director of Laboratories, The Walker-Gordon Laboratory Co., Inc., Plainsboro, N. J.

IN THE WORK of controlling Bang's disease, it has been emphasized repeatedly that the success of eradication measures, based on the detection and subsequent disposal of infected animals, depends to a great extent upon the animals brought in to replace the diseased ones that are slaughtered. This is true whether the control measures are being applied to a herd or on an area basis.

Other things being equal, the degree of self-containment of a herd or of an area determines the ease with which Bang's disease infection may be rooted out and kept out. Herds in which replacement requirements are limited usually can be replenished satisfactorily from healthy home-bred stock or from purchased animals, carefully selected and tested. Large herds, especially those operated under high standards of health and economic production, are subjected to much greater stress in obtaining replacements, both as to quality and quanity. The stress of these replacements, whether purchased or home-bred, inevitably leads to complications and prolongs the struggle to free the herds and keep them free from Bang infection.

The purchase of cows negative to the agglutination test for Bang's disease is not such a problem now as it was ten, or even five, years ago. However, the negative cow purchased, if she is really noninfected and from a noninfected herd, is apt also to be nonresistant. Such nonimmunes are put at risk if added to a herd that is not entirely free of infection. On the other hand, the negative cow, originating from an infected herd, may endanger the herd to which she is added. In either case, the hazard is obvious. In spite of the great acceleration of Bang's disease testing and herd accreditation work since 1934, there are still not a sufficient number of herds to serve as an economic source of large numbers of desirable replacements that are dependably free from Bang infection.

In the case of home-bred heifers, these serve as a desirable source of additions when raised free from Bang's disease and when added to herds that are likewise free from that infection. However, the high susceptibility of pregnant heifers to virulent Bang infection is well recognized. Such highly susceptible animals suffer severe losses when added to herds that contain but a small amount of *Brucella abortus* infection and they keep the infection alive indefinitely.

It is this pronounced susceptibility of the heifers added which has led us to study the possibilities of conferring some degree of resistance by vaccination. Well bred, properly raised heifers have been demonstrated to possess several advantages as replacements, especially in herds devoted to the economic production of quality milk, in which standards of herd health are necessarily high. The breeding and health records of the heifers are known; they present a minimum of udder troubles when admitted to production; their best productive years are available to the owner.

Among the numerous investigations on vaccinal immunity to the abortion phase of Bang's disease, two are of fundamental interest with respect to the use of vaccines in young bovines.

The first, begun by Theobald Smith and Ralph B. Little, of the Rockefeller Institute, in 1917 and reported in 1923[1] is a thorough study of the effect of cultures of *Br. abortus* on the bovine species. The studies of Smith and Little related principally to living strains of *Br. abortus* having "a considerable degree of virulence" for guinea pigs and used as a vaccine in virgin heifers of about breeding age. They also used multiple inoculations of heat-killed

*Presented at the seventy-fifth annual meeting of the A. V. M. A., New York City, July 5-9, 1938.

cultures. The generally favorable results obtained by the use of both the living and heat-killed cultures led these investigators to make the following conclusions, among others, which are pertinent to our present consideration of calfhood immunization.

> Vaccination with small doses of living bacilli (one agar slant or less), practiced two to three months before conception, is not a dangerous procedure.
>
> A small proportion of virgin heifers have a high blood titre and they may be regarded as infected, the virus being localized in the udder.
>
> The relative efficiency and danger of recently isolated strains of *Bacillus abortus* and those under cultivation for months and years when used as vaccines is not clear. Under the circumstances older cultures should be used.
>
> There is probably no gain in two injections of living vaccine, since high agglutinin titres of the blood are established by single injections and in a fair proportion maintained during several pregnancies.
>
> The use of vaccine does not in itself tend to eliminate the infectious agent from any herd, although it may greatly reduce the infectious material quantitatively by reducing the number of cases of placental disease.

The second investigation or, rather, series of investigations to be mentioned is that conducted by Cotton, his late associate, Buck, and their coworkers in the U. S. Bureau of Animal Industry. These studies also were begun in 1917 and have been reported on a number of occasions.[2-8] These reports are so recent and have been the subject of so much current interest and discussion that a summary of them is unnecessary.

It should be noted that Smith and Little employed rather virulent strains of *Br. abortus* in most of their work; they also vaccinated animals approaching breeding age. As a result, the vaccinated animals, as already noted, developed high agglutinin titres of the blood which were inclined to persist. Hence, their study of vaccination against Bang's disease, while contributing much fundamental knowledge, did not immediately lend itself to the development of a method of immunization compatible with a blood-testing program.

On the other hand, the calfhood vaccination method developed by Cotton and his associates requires cultures of low virulence applied at an early age. The resulting agglutinin production is relatively temporary which, in so far as is known, makes the method compatible with the existing control programs based on blood tests and elimination of reactors.

This factor, and other apparent advantages of calfhood vaccination, led us to undertake in 1935 a field trial of the method with the coöperation of Dr. Cotton and his associates at the Animal Disease Station of the federal Bureau of Animal Industry and with the permission of the New Jersey Bureau of Animal Industry. Our herds have operated since 1928 under a strict blood-testing, control program to eliminate Bang's disease. The replacement requirements in herds such as ours are heavy, as already noted. In spite of repeated blood tests and the application of sanitary measures, some residual Bang infection still exists in our herds. Heifer replacements are desirable but not economical unless they can be given some degree of resistance to even the slight exposure to which they may be subjected after entering the producing herds. The trial of calfhood vaccination was looked upon not as a substitute for other methods of control, but simply as an aid in combating Bang's disease. It was felt that a gain would be made if, as suggested by Smith and Little, vaccination would "reduce the infectious material quantitatively by reducing the number of cases of placental disease."

Beginning in May, 1935, various groups of heifers have been inoculated with Strain 19 vaccine prepared and furnished by the federal Bureau of Animal Industry.* The data here reported relate to some of the observations that have been made with respect to:

1. Systemic effects of the vaccine.
2. Effect of the vaccine injections upon agglutinin titre.
3. Results of first pregnancies in heifers vaccinated in 1935.
4. Disposal losses on account of positive blood reactions in vaccinated and control heifers.

*The first two groups inoculated in 1935 were treated with a commercial preparation of Strain 19.

TABLE I—Study of agglutination test behavior of 625 heifers following vaccination with Strain 19.

Breed	Number	Per Cent
Holsteins	234	37.4
Guernseys	307	49.1
Jerseys	24	3.8
Brown Swiss	60	9.7
Totals	625	100.0

Age Distribution at Time of Vaccination

Months	Holsteins	Guernseys	Jerseys	Brown Swiss	Number	Per Cent
4	39	45	2	9	95	15.2
5	45	82	3	18	148	23.7
6	63	91	6	11	171	27.3
7	52	54	6	8	120	19.2
8	31	27	3	12	73	11.7
9	4	8	4	2	18	2.9
Totals	234	307	24	60	625	100.0

70.2 per cent of heifers 5-7 months of age.

TABLE IA—The mean agglutination titres of 25 heifers following a single inoculation of Brucella vaccine Strain 19 (heifers 5-8 months of age).

Interval After Vaccination	Dilutions								
	1:25	1:50	1:100	1:200	1:400	1:800	1:1600	1:3200	1:6400
*	—	—	—	—					
10 days	+	+	+	+	+	+	I	I	I
20 days	+	+	+	+	+	I	I	I	—
1 month	+	+	+	I	I	—	—	—	—
2 months	+	+	I	I	I	—	—	—	—
3 months	+	I	I	I	I	—	—	—	—
4 months	I	I	—	—					
5 months	I	I	—	—					
6 months	—	—	—	—					

*Prior to vaccination.

TABLE II—Agglutination test behavior of 625 heifers vaccinated with Strain 19.

Breed	Heifers	Returned to Complete Negative		Continued to React Slightly or Intermittently		Continued Positive	
		Number	Per Cent	Number	Per Cent	Number	Per Cent
Holsteins	234	185	79.0	49	20.9	0	0.0
Guernseys	307	245	79.8	61	20.0	1	0.1+
Jerseys	24	22	91.7	2	8.3	0	0.0
Brown Swiss	60	52	86.7	8	13.3	0	0.0
Totals	625	504	80.6	120	19.2	1	0.1+

TABLE III—Age at vaccination in relation to time required for 504 heifers to return to negative agglutination status.

Age at Vaccination	Heifers	Returned to Negative Status in—							
		3 Months		6 Months		9 Months		12 Months	
		Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
4 months	93	26	28.0	62	66.6	5	5.4	0	0.0
5 months	131	25	19.1	91	69.4	10	7.6	5	3.8
6 months	141	8	5.7	94	66.6	31	22.0	8	5.7
7 months	85	11	13.0	52	61.2	15	17.6	7	8.2
8 months	44	0	0.0	29	65.9	7	15.9	8	18.2
9 months	10	4	40.0	3	30.0	3	30.0	0	0.0
Totals	504	74	14.7	331	65.7	71	14.1	28	5.5

Systemic Effects of the Vaccine

We have seen no ill effects from the inoculation of Strain 19 vaccine in some 1,200 heifers in so far as their physical well-being is concerned. All inoculations have been made under the skin, midway of the neck, on the right hand side, and with the usual precautions. A local swelling about the size and thickness of the hand develops after two or three days. These swellings disappear in ten days to two weeks. In only one instance have we seen evidence of suppuration, an orange-sized swelling which underwent resorption without opening. Few heifers go off feed. We have taken the temperatures of a few heifers following vaccination and find that there is a rise of two to four degrees above normal, beginning within 24 hours after the inoculation and lasting for three or four days.

Effect of Vaccine Injections on Agglutinin Titres

In milk-producing herds that must be tested continuously to eliminate any animals infected with Bang's disease, no treatment should be applied that will obscure or confuse the diagnostic reactions. Although it had already been established that the inoculation of Strain 19 into calves of suitable age had no permanent effect upon subsequent tests for Bang's disease, it was interesting to note the periods of time necessary for vaccinated animals to develop their maximum titres and then to return to a negative status. For this information we have studied the records of 625 heifers which were retested with sufficient regularity after vaccination to furnish a fairly accurate picture of this phase. The results are summarized in tables I to V, inclusive.

Table I gives the breed and age distribution of the 625 animals whose agglutination records are analyzed in tables II, III and IV.

It will be noted that 70 per cent of the 625 heifers were within the age limits (5 to 7 months) usually recommended for the use of Strain 19 vaccine; an additional 15 per cent were below that age. If eight months is considered the extreme age at which calfhood vaccination should be employed, then only about 3 per cent of the animals included in this summary exceeded that limit.

Table IA depicts what may be taken as an average agglutination response for the calves receiving a single injection of Strain 19 vaccine. Only a small number of all the heifers inoculated were tested at ten- and 20-day intervals in order to determine the time of maximum agglutinin titre. Table IA is a cross section of the tests on 25 such heifers.

It will be seen that a "high" of 1:6,400 was reached in about ten days after vaccination; after this time there was a gradual decline and the "low," or completely negative status, was reached in about six months. There were some wide individual variations in this respect, as might be expected. Some animals developed titres as high as 1:12,800 and 1:25,600, but these

were exceptional. Also, some heifers which apparently possessed poor antigen-antibody reaction mechanisms did not begin to react until two or three weeks following injection and then reached a maximum of only 1:200 or 1:400.

Table II summarizes the agglutination test behavior of the 625 vaccinated heifers as to breed and according to whether they returned to a completely negative status, continued to react slightly, or remained positive.

TABLE IIIA—Summary of table III.

	Number	Per Cent
Returned to negative status	504	80 6
Continued to react slightly	120	19 2
Remained positive	1	0 2
Totals	625	100 0

TABLE IV—Age at vaccination in relation to 120 heifers that continued to react slightly or intermittently.

Age at Vaccination	Number of This Age Vaccinated	Retained Partial Agglutination Titres (1:50 or Less)	
		Number	Per Cent
4 months	95	2	2 1
5 months	148	17	11 5
6 months	171	30	17 5
7 months	120	34	28 3
8 months	73	29	39 7
9 months	18	8	44 4
Totals	625	120	19 2

TABLE V—The mean agglutination titres of 19 heifers following multiple inoculations of Brucella vaccine—Strain 19 (all heifers 4 to 5 months of age at first vaccination).

Date	Dilutions									Remarks
	1:25	1:50	1:100	1:200	1:400	1:800	1:1600	1:3200	1:6400	
7-27-37	—	—	—	—						Before vaccination
7-27-37										1 cc. Strain 19 (7-13-37) s. c.
8- 4-37	+	+	I	—	—	—	—	—	—	3 cc. Strain 19 (7-2-37) s. c.
8-10-37	+	+	+	+	+	I	—	—	—	5 cc. Strain 19 (8-3-37) s. c.
8-17-37	+	+	+	+	+	+	I	—	—	
8-24-37	+	+	+	+	+	I	—	—	—	
8-31-37	+	+	+	+	+	—	—	—	—	
9- 8-37	+	+	+	+	—	—	—	—	—	10 cc. Strain 19 (8-17-37) s. c.
9-15-37	+	+	+	+	+	I	—	—	—	
9-22-37	+	+	+	+	+	I	—	—	—	
9-30-37	+	+	+	+	I	—	—	—	—	
10- 9-37	+	+	+	I	—	—	—	—	—	
10-27-37	+	+	+	—	—	—	—	—	—	
1-15-38	+	I	—	—	—	—	—	—	—	
2-26-38	+	I	—	—	—	—	—	—	—	
4- 7-38	+	—	—	—						
6- 7-38	I	—	—	—						

TABLE VI—Results of first pregnancies in heifers vaccinated in 1935.

Date Vaccinated	Treatment	Pregnancies Terminated	Normal Full-Term Calves		Abortions (All Kinds)		Abortions (Brucella Infection)	
			Number	Per Cent	Number	Per Cent	Number	Per Cent
5- 8-35	9 vaccinated	8	7	87.5	1	12.5	0	0.0
	7 controls	6	6	100.0	0	0.0	0	0.0
7-30-35	12 vaccinated	11	11	100.0	0	0.0	0	0.0
	2 controls	2	2	100.0	0	0.0	0	0.0
5-14-35	7 vaccinated	6	5	83.3	1	16.7	1	16.7
	11 controls	11	10	90.9	1	9.1	1	9.1
6- 4-35	35 vaccinated	32	31	97.0	1	3.0	0	0.0
	23 controls	23	23	100.0	0	0.0	0	0.0
7-22-35	15 vaccinated	6	6	100.0	0	0.0	0	0.0
	16 controls	10	7	70.0	3	30.0	3	30.0
10- 9-35	14 vaccinated	13	12	92.3	1?	7.7	0	0.0
2-12-36	6 controls	4	4	100.0	0	0.0	0	0.0
10-25-35	12 vaccinated	11	10	90.9	1?	9.1	0	0.0
	2 controls	2	2	100.0	0	0.0	0	0.0
10-28-35	11 vaccinated	11	11	100.0	0	0.0	0	0.0
	2 controls	2	2	100.0	0	0.0	0	0.0
11- 7-35	28 vaccinated	26	24	92.7	2	7.3	2?	7.3
	4 controls	4	4	100.0	0	0.0	0	0.0
Totals	143 vaccinated	124	117	94.4	7	5.6	3	2.4
	73 controls	64	60	93.8	4	6.2	4	6.2

It will be noted that 504, or about 80 per cent, of the heifers reached a negative status and that 120, or about 20 per cent, continued to react slightly or intermittently. By "negative" is meant no agglutination in either the 1:25, 1:50, 1:100 or 1:200 dilutions used in the routine tests. By slight reactions are meant partial or sometimes complete reactions in the 1:50 dilution, the latter accompanied by a partial (1+) reaction in the 1:100 dilution. Only one heifer in this series continued as a "positive" or complete reactor; she was nine months of age at the time of vaccination.

In table III is shown the time required for the 504 heifers to return to a negative status in relation to their age at vaccination. As previously noted, a majority (in this instance 80.4 per cent) of the heifers that became completely negative after vaccination with Strain 19 did so within six months. With these animals, age is apparently not a factor.

In the case of heifers that required more than six months to return to negative, there was a greater percentage of such animals in the higher age brackets (7 to 9 months) than in the lower (4 to 6 months).

Table IV is an analysis of the age factor in relation to the 120 heifers in the group that continued to react slightly or intermittently for indefinite periods after vaccination.

In this group, there is a definite correlation between age and tendency to maintain some response to agglutination tests. A greater proportion of the heifers in the upper age brackets at the time of vaccination retained partial agglutination titres indefinitely.

It is believed that these partial titres are of little significance with respect to evidence of permanent "infection" with Brucella organisms contained in the Strain 19 vaccine. Repeated agglutination tests have been made on the milk sera of such heifers after freshening. To date, none has given

any indication by this method that the undeveloped udder may have served as a permanent focus for the organisms. Also, through the coöperation of Dr. H. J. Metzger at the New Jersey Agricultural Experiment Station, milk samples from a number of such slightly reacting vaccinated heifers have been inoculated into guinea pigs with entirely negative results. These experiments will be reported elsewhere.

Multiple Inoculations

Table V illustrates the effect of repeated inoculations of Brucella vaccine upon the agglutinin titres of a group of heifers, which received four injections of Strain 19; the second, third and fourth injections were made one, two and six weeks, respectively, after the first dose was given.

Smith and Little, as previously quoted, concluded that there is probably no gain in two injections of living vaccine, since single injections caused high agglutinin titres that were maintained in a fair proportion of their heifers during several pregnancies. They worked with more mature heifers than is now the practice with calfhood vaccination. The tendency of such older animals to acquire persistent agglutinin titres after administration of living cultures is recognized.

In young calves, the efficiency of the immunity-producing mechanism is perhaps less certain than in older animals. For this reason, it was decided to determine the effects of more than one inoculation of Strain 19 vaccine in such animals. Two groups of heifers have been so treated to date.

In the first group (see group VI, table VI), the heifers were 4 to 5 months of age when first vaccinated and 8 to 9 months of age when revaccinated. Of the 14 heifers so treated, nine have shown a definite tendency to maintain a partial agglutinin titre in the 1:25 or 1:50 dilution; the remaining five returned to complete negatives. The first pregnancy records on these heifers have been very satisfactory. Five cc. was used for the first dose on October 9, 1935, and 10 cc. for the second dose on February 12, 1936.

For the second group receiving multiple inoculations, 19 heifers four to five months of age were selected and treated as shown in table V; 21 additional heifers six to seven months of age received one injection only for comparison. These animals will not be due to calve until some time next year.

The effect of the multiple inoculations on the average agglutinin titre of the 19 heifers, as shown in table V, was not as pronounced as anticipated. The heifer which reacted most strongly reached a titre of 1:6,400 two weeks following the first injection of 1 cc. of vaccine and one week after the second injection of 3 cc. The third inoculation of 5 cc. caused no increase over the previous titre. Following the fourth injection of 10 cc. of vaccine (given one month after the third dose), this heifer's titre, which had fallen to 1:400, rose only to 1:800. Table V represents the average titres maintained by the 19 heifers.

First Pregnancies in 1935 Series

Most of our heifers are bred to calve at about 2½ years of age. The data on pregnancies in this vaccination experiment will be confined in this report to the first preg-

TABLE VII—Disposals on account of positive agglutination reactions in vaccinated and control heifers.

Group	Heifers	Reacted Before Calving		Reacted Since Calving		Total Disposals on Account of Positive Reactions	
		Number	Per Cent	Number	Per Cent	Number	Per Cent
Vaccinated	143	6	4.2	8	5.6	14	9.8
Controls	73	6	8.2	9	12.3	15	20.5
Totals	216	12	5.6	17	7.8	29	13.4

nancies only of the 1935 series of heifers. The data on the second pregnancies of these animals are not complete enough for analysis at this time.

Table VI presents a summary of the first pregnancies that have been terminated in nine groups of vaccinated and control heifers comprising the 1935 series. All of the animals vaccinated in these groups received one injection only, with the exception of group VI, which was re-vaccinated as previously stated. All groups, except I and II, were treated with Strain 19 vaccine supplied by the federal Animal Disease Station at Beltsville, Md. Groups I and II were treated with a commercial preparation of Strain 19.

Of the 143 vaccinated and 73 control animals comprising the 1935 totals, 124 of the former, or 86.7 per cent, and 64 of the latter, or 87.7 per cent, had terminated their first pregnancies at the time this report was prepared. Of the vaccinated group, 117, or 94.4 per cent, gave birth to normal full term calves; of the controls, 60, or 93.8 per cent.

As shown in the table, abortions occurred in both the vaccinated and control groups. In the vaccinated group there were seven, or 5.6 per cent, out of 124 terminated pregnancies. Of these total abortions, only three, or 2.4 per cent, were apparently caused by Brucella infection.

In the control group there were four abortions, or 6.2 per cent of 64 terminated pregancies; of these, all four were apparently due to Brucella infection.

Of the three vaccinated heifers, which are designated as aborters due to Brucella infection (one in group III and two in group IX), the one in group III developed a strongly positive blood titre in her fifth month of pregnancy. She dropped a living calf at 253 days and the placenta was passed normally. There is some doubt about this heifer's actually being an aborter, although she is so classed in our data.

Of the two vaccinated heifers in group IX, which are designated as aborters due to Brucella infection, one aborted at the fifth month and developed an agglutninin titre of only 1:50. This titre remained for four months and the animal was sent to slaughter for other reasons. The second heifer also aborted at the fifth month; three weeks later her agglutinin titre was only partial in 1:50. This animal also was sent to slaughter.

In the four abortions occurring in the control group, all were ascribed to Brucella infection because of positive reactions to the agglutination test at the time the abortions occurred.

Evidence as to whether the Bang organism was the causative agent of the abortions in the first pregnancies of both vaccinated and control animals in the 1935 series had to be based on subsequent agglutination tests, since facilities were not available for guinea pig inoculations. Since that time, arrangements have been made for diagnostic inoculations and such examinations are now being made whenever possible in the case of abnormal calvings.

DISPOSAL LOSSES

The herds in which this experimental work is being done operate under regulations which require that the producing animals shall be maintained free from Bang reactors as determined by repeated blood tests. Consequently, any vaccinated or control heifers which develop positive blood titres either before or after calving are ineligible for admission to the producing herds.

It is realized that the vaccination of calves does not control their subsequent blood test behavior, *i.e.*, prevent their reacting again to the agglutination test if they are exposed to Brucella infection. We have studied the blood-test records of all the heifers in the 1935 series and noted those that have reacted to date. These are shown in table VII. Fourteen, or 9.8 per cent, of the 143 vaccinated animals have been disposed of on account of positive blood titres since the experiment started; 15, or 20.5 per cent, of the control animals have been disposed of for the same reason. These figures include the positive aborters.

These disposals, which are in excess of normal experience, were caused principally by one group (group V) consisting of 15 vaccinated heifers and 16 controls. A con-

trol heifer in this group aborted at pasture, in the summer of 1937, and infected a considerable number of the animals in the group. This aborter was found to be positive to the blood test. As a result of this abortion and subsequent infection, seven other control heifers became positive and two of them aborted; the other five had to be disposed of before calving. In addition, ten of the vaccinated heifers in the same group subsequently reacted. Four of these were held until their pregnancies were terminated with no abortions; the remaining six had to be disposed of before calving.

If the heifers in group V are excluded from the results shown in table VII, we find four heifers disposed of because of positive blood reactions in 128 vaccinated animals, or 3.1 per cent. For the controls, the disposals would be seven out of a total of 57, or 12.3 per cent.

SUMMARY

1. Following the subcutaneous administration of Strain 19 Brucella vaccine to calves, no serious systemic effects were observed. There was local swelling and some elevation of temperature, but both of these effects were transient and subsided without incident.

2. Single injections of the vaccine caused a pronounced agglutinin response which usually reached a peak in about ten days and then gradually diminished. In 80 per cent of 625 vaccinated heifers whose agglutinin titres were studied, the test became completely negative within four to six months. In about 20 per cent, a low titre of 1:50, or partial in 1:100, was maintained indefinitely. These low titres have not been found to be associated with any permanent "infection" with the Strain 19 culture. Only one heifer in the group of 625 retained a positive blood test indefinitely.

3. In 124 first pregnancies in vaccinated heifers, there were seven abortions, or 5.6 per cent. Only three of these abortions were ascribed to Brucella infection, or 2.4 per cent of the pregnancies. In 64 first pregnancies in control heifers, there were four abortions, or 6.2 per cent, all of which were apparently due to Brucella infection.

4. A number of heifers in the 1935 series developed positive blood titres due to natural exposure to infection. The disposals on this account, including positive aborters, in the vaccinated group amounted to 9.8 per cent, and in the control group to 20.5 per cent.

References

[1]Smith, T., and Little, R. B.: Studies in vaccinal immunity towards diseases of the bovine placenta due to *Bacillus abortus* (infectious abortion). Rock. Inst. Med. Res., Mono. 19 (1923), p. 124.

[2]Buck, J. M., and Creech, G. T.: Studies and observations relating to the immunology of bovine infectious abortion. Jour. Agr. Res., xxviii (1924), 7.

[3]Buck, J. M.: Studies of vaccination during calfhood to prevent bovine infectious abortion. Jour. Agr. Res., xli (1930), 9.

[4]Cotton, W. E.: Efficacy of different strains of *Brucella abortus* as immunizing agents against infectious abortion. Jour. Agr. Res., xlv (1932), 12.

[5]Cotton, W. E., Buck, J. M., and Smith, H. E.: efficacy of an avirulent strain of *Brucella abortus* for vaccinating pregnant cattle. Jour. Agr. Res., xlv (1933), 4.

[6]Cotton, W. E., Buck, J. M., and Smith, H. E.: Studies of five *Brucella abortus* (bovine) strains as immunizing agents against Bang's disease (infectious abortion). Jour. A. V. M. A., lxxxv (1934), n. s. 38 (2), pp. 232-247.

[7]Cotton, W. E.: Vaccination against Bang's disease. Report of Atlantic States Conference on Bang's Disease (1937).

Discussion

DR. B. T. SIMMS: There was one group mentioned in which I was particularly interested. I did not get the full significance of the results in that group because the figures at the end of the discussion applied to the entire group. As I understand it, you have one group of control and vaccinated animals in a pen or pasture in which there were abortions. That is the type of condition that will occur on the farm. Now, I would like to have those two groups segregated. How many were vaccinated and how many control animals were there that you know were infected? What were the results?

DR. HARDENBERGH: There were 15 vaccinated animals and 16 controls. These 31 heifers were all turned out on pasture together. The abortion occurred in the summer of 1938, in a control heifer. She was found to be positive to the blood test. As a result of that abortion, seven other control heifers became positive. In other words, that made a total of eight out of the 16 controls that became positive. Counting the first abortion in a control animal, two other control heifers aborted and were positive, of course. In other words, three out of the total of eight positives actually aborted. We had to dispose of the five others. I could not get

the man to hang on to them. He detested them because they were positive. He refused to keep them on the place to see whether they would terminate their pregnancies. With the 15 vaccinated heifers, ten of those became positive to the blood test. We kept four of them and not one aborted. The six others were disposed of, but we do know that four vaccinated heifers, positive, due to natural exposure, carried their calves to full term.

DR. C. H. CASE: How often were the blood tests made on the heifers? Every month or every two months?

DR. C. A. BRANDLY: What was the age of these heifers that were exposed naturally—the controls as well as those vaccinated?

DR. W. E. COTTON: I would like to say that I believe Dr. Buck and myself were very much pleased to have Dr. Hardenbergh undertake this experiment, which is a rather large field experiment with laboratory control. We feel that it is going to bring some results. I would like to be kept informed as to the results, Dr. Hardenbergh.

DR. C. P. FITCH: I would like to ask if you have the milk titres on the group of vaccinated animals which were held in this small group of which Dr. Simms spoke?

DR. HARDENBERGH: Dr. Case asked how often these heifers had been retested. The 625 heifers which were included in the summary presented were retested with considerable frequency. Whenever possible, we retested them—as often as every month. Some of our heifers are shipped away to young-stock farms where they may be tested every three months. In the case of some sent out to your state, we do not get tests on them quite as frequently as that. We do plan to test all of these heifers frequently, after they are bred, because we want to keep track of them very clearly from the time they are bred. Thus, starting at about 20 months of age, all of these animals are tested every month, if possible.

Dr. Brandly asked the age of the heifers that aborted in the particular group referred to. All the animals in that group were vaccinated between five and eight months of age. The control animals and the vaccinated animals were nearly the same age. There might be a difference of a month or two between some, but most of them averaged between five and eight months.

This experiment is disappointing to us for the reason that we did not arrange for more careful laboratory examination of every fetus dropped. Unfortunately, some of them were dropped in the pasture where these heifers were not under close observation, and the abortions were discovered too late to obtain either the fetus or placenta, but we hope to correct that condition.

Dr. Fitch asked about the milk-serum titres. We did not make any tests of milk-serum titres on those animals.

Vaccination for Bang's Disease

The United States Bureau of Animal Industry disapproves by regulation the use of live-culture vaccine in adult cattle. . . . It does neither harm nor any good and may condemn as infected vaccinated animals offered for sale. . . . Owners not informed of this fact have a just cause for legal action against the veterinarian who does the vaccinating. . . . Dishonest owners have cows vaccinated to have them condemned as infected animals in order to collect the indemnity. . . . The vaccination of adult cattle should be frowned upon. . . . In a series of experimental vaccinations of calves reported by the Bureau, 88 per cent went safely through two pregnancies; the remaining 12 per cent were not protected. —*C. R. Donham, before the Iowa Veterinary Medical Association.*

Staphylococcic Infections

Veterinary medicine of the past appears to have overlooked the true pathogenic rôle of the genus Staphylococcus in the family of bacteria. The genus Streptococcus, on the contrary and obviously not without reason, has been kept uppermost in mind. Recent investigational work on mastitis of cows and bitches and on various dermatoses, of which eczema and follicular mange of dogs are examples, seem, however, to indicate that the staphylococci disseminated over the surface of the body are ever ready to surmount the dermic barrier and cause troubles heretofore unappraised. The various types of acne, furuncles, phlegmons and staphylococcemia make up a group of ailments worthy of considerable attention, if current reports may be taken as criteria.

In an observation made on 264 cows slaughtered in an abattoir in Bucharest, trichomonads could not be found in the uterus of any healthy cow nor in the uterus of heifers under two years of age. Out of 15 uteri affected with metritis, the parasite was found in nine, or 60 per cent.

Observations on the Stiff Lamb Problem with Special Reference to White Muscle Disease*

By L. R. VAWTER, D. V. M., and EDWARD RECORDS, V. M. D.
University of Nevada, Reno, Nev.

THE TERM "stiff lambs," as applied by sheepmen, is a collective term actually embracing several separate diseases and infections such as those caused by *Actinomyces necrophorus, Corynebacterium pyogenes, Erysipelothrix rhusiopathiae, Clostridium tetani,* and "white muscle" disease. Regardless of the nature of these diseases, one of the first symptoms noticed by sheepmen is lameness or stiffness.

While necrophorus infection may manifest itself in several forms in sheep, perhaps the two most commonly observed necrophorus infections are liver rot and foot rot. Liver rot, characterized by multiple, grayish, nodular, necrotic areas in the liver of young lambs, is believed to be contracted soon after birth by way of the navel. It may cause heavy losses in the first few weeks after lambing. Although the liver is perhaps the common primary point of localization, metastasis and contiguity may give rise to a variety of symptoms and lesions. One outbreak of necrophorus infection is recalled among young lambs varying in age from two to six weeks wherein stiffness and apparent lameness of the front legs was traceable to extensive fibrinous pleuritis and pneumonia associated with this infection. We have not observed foot rot in young lambs.

The "stiff lambs" appearing among feeder lambs four to six months old may be attributed to *A. necrophorus, C. pyogenes,* or *E. rhusiopathiae* infections. Necrophorus infection in lambs of this age is likely to be localized in the interdigital spaces, causing severe local suppurative necrosis accompanied by lameness or stiffness. Extension of the infection to articular surfaces is not uncommon. Extensive visceral lesions such as occur in lambs a few weeks old have not been observed in lambs of feeder age.

In recent years *E. rhusiopathiae* has been found associated with polyarthritis and stiffness among feeder lambs. In 1937 we examined lambs from a band of 3,500 feeders, of which about 1,000 head showed marked symptoms of stiffness and loss of condition soon after arrival in the feed lot. The lambs came from three widely separated ranches. Among those from one ranch, *E. rhusiopathiae* appeared to be the infection responsible for the polyarthritis and stiffness, while among those from another ranch, *C. pyogenes* appeared to be responsible. Stiffness and arthritis, with swelling involving the joints of the legs and coxofemoral articulations, were common to both infections. The identity of the organisms could not be established on the basis of physical examination. In the lambs having *C. pyogenes* infection, multiple abscesses occurred in the liver and lungs. These were not observed in the group where *E. rhusiopathiae* was recovered from affected joints and appeared to be responsible for the arthritis.

The time and place where these infections were first contracted were not established, as all of the lambs were born and reared under semi-range or open-range conditions. From the extent and character of the lesions associated with these infections it was presumed that they had been smoldering for an indefinite period, suddenly flaring up and coming to clinical notice following such incidents as sorting, shipment, and change of feed and environment. According to the observations of Marsh,[1] swine erysipelas infection may cause arthritis in lambs one week old.

Tetanus occurs rather frequently among young lambs in small ranch flocks as well as in large range bands. Docking and cas-

*Published with the approval of the Director of the Nevada Agricultural Experiment Station in connection with Research Project No. 39. Presented at the annual meeting of the Intermountain Livestock Sanitary Association, Salt Lake City, Utah, January 9-11, 1939.

trating are usually performed in the intermountain country when the lambs are one or two months old. Some rather heavy losses from this infection come to our attention almost every year. One of the heaviest was in a range band of lambs where tetanus was contracted on an old lambing ground that had been used about 40 years without any sanitary precautions whatsoever. Stiffness is the prominent early symptom, but generalized muscular tetany and opisthotonus follow in rapid order.

"White muscle" disease, also called white flesh, is a disease of young lambs three to ten weeks old. The majority of cases in this region have been observed among "hot house" lambs or those born in early or midwinter, when unfavorable weather requires the confinement of ewes and lambs in pens or sheds on a more or less restricted diet and exercise. We have observed this disease only in small ranch flocks and never among lambs born and reared on the open ranges or under semi-range conditions. It may occur under range conditions but, if so, the loss has been so low that it has escaped notice. The breed of lamb apparently bears no relationship, as it may occur among lambs born of rugged cross-bred range type ewes as well as those from pure-bred animals. Genetic factors do not appear to play any part.

The first symptom noticed is stiffness followed in a day or two by muscular weakness and prostration. There is no febrile reaction during the early part of the disease. Affected lambs may actually starve to death by reason of their inability to stand up and nurse. While the disease may be suspected, positive diagnosis can be made only by autopsy. Up to this time we, as well as others, have been unable to incriminate any bacterial pathogens as the cause of "white muscle" disease of lambs. From the work of Hagan and Metzer[2] and Willman,[3] muscle damage undoubtedly is present some time before symptoms of stiffness appear.

The factors contributing to the occurrence of this disease of young lambs are still obscure. Marsh, in 1932, suggested the possibility that improper calcium-phosphorus ratio may have some relationship to this disease. This contention is partially supported by the work of Willman and coworkers. Willman reported that "white muscle" disease occurred among lambs in a group of ewes receiving a diet of oats, barley, cull beans, and alfalfa hay. Stiff lambs did not occur in the groups receiving a diet of oats, wheat bran, corn silage, and mixed hay, which is considerably higher in phosphorus than that received by the stiff lamb group.

In contrast to Willman's results, we recently observed a small ranch flock of ewes and lambs confined in pens wherein stiff lambs, or "white muscle" disease, occurred only in the group receiving alfalfa hay alone. No stiff lambs occurred among another group of ewes receiving alfalfa hay from the same stack and rolled oats. Alfalfa hay in the vicinity of Reno generally runs somewhat lower in phosphorus than in other parts of the country or in some other parts of western Nevada. Rolled oats is a fair to good source of phosphorus, although inferior to wheat bran. Three affected lambs recovered in about two weeks after being turned out with the ewes on dry alfalfa stubble in the early spring and upon addition of rolled oats to the diet of the ewes. It is possible that range or semi-range conditions afford a better opportunity for the ewes to balance their diet than would be the case under pen conditions and restricted diet.

Hobmaier[4] pointed out that the muscle changes in lambs correspond entirely with those observed in horses affected with azoturia. We frequently observe azoturia among ranch horses that have received unrestricted amounts of alfalfa hay alone during a period of idleness, whereas it has not been observed among other horses in the same group receiving the same food but in the meantime doing some work which would require a higher plane of metabolism.

In 1921 and 1922, Dr. Stephen Lockett and the writer observed a disease of young calves which closely resembled "white muscle" disease of lambs. The extensive bilateral muscle lesions were very similar, if not identical, in gross and histological

appearance to those observed in lambs. The calves were being raised from birth on a diet consisting mainly of skimmed milk. Oatmeal gruel appeared to be beneficial when added to the diet of affected calves before the disease became too far advanced. There is some indication in this instance as well as that reported with lambs that the vitamin B complex found in oats may have some beneficial action both in prevention and cure of this disease.

Hjarre and Lilleengen[5] recently reported that they were able to reproduce the degenerative muscle changes (white flesh) in experimental calves using a diet deficient in vitamins B and C. Madsen[6] and coworkers observed that while sheep on synthetic diets required some alcohol-soluble factor found in the vitamin B complex of yeast, they were able to raise sheep from weaning time to maturity on synthetic diets devoid of vitamin C.

Bechdel[7] and associates found that bacterial activity in the paunch of the cow apparently plays an important rôle in the synthesis of vitamin B and, therefore, the low intake requirements of bovidae for vitamin B may be accountable to this phenomenon. It is presumed that a similar situation may prevail in other ruminants, particularly sheep.

There appears to be a missing link in our knowledge concerning the requirements of vitamins B and C for young ruminants, such as lambs and calves, during the period when they subsist almost entirely on milk either from the dam or are fed artificially. Milk alone is not regarded as an adequate source of either of the vitamins B or C for some species. "White muscle" disease of young lambs and calves appears at an age when the rumen and accessory structures are still rudimentary and certainly not fully capable of assuming the physiological rôle that they play later in life, in addition to being a synthetic source of vitamin B and possibly C. As suggested by the work and observations of others as well as our own, either nutritional or metabolic factors, possibly a combination of both, may contribute to the occurrence of "white muscle" disease.

As now used by sheepmen and others, the term "stiff lamb" disease is an indefinite one embracing a variety of pathological conditions peculiar to lambs. Stiffness and muscular weakness occurring in lambs two weeks to three months of age may be due to necrophorus, swine erysipelas, or tetanus infections, and "white muscle" disease. The latter is still obscure in etiology.

The infectious diseases are traceable to unsanitary conditions in the lambing sheds or lambing beds on the open range and careless surgical technic. Some of them, such as swine erysipelas and *C. pyogenes* infections, may smolder unnoticed and come to clinical attention when the lambs are of feeder age. "White muscle" disease has not been observed in this locality among lambs above ten weeks to three months of age.

From observations of the writer, all of the diseases included under the category of stiff lambs are preventable if proper attention is given to sanitary precautions and methods of feeding and management of the ewes both prior to and after lambing. Treatment and remedial measures instituted after any of the enumerated diseases appear in a band of lambs has not proven wholly satisfactory. Accurate diagnosis can be made only after careful physical, postmortem, and bacteriological examination in connection with these diseases coupled with a knowledge of the methods of management, feeding, and history of the premises in regard to lurking infections which may not always manifest themselves in other species of live stock.

References

[1]Marsh, H.: Jour. A. V. M. A., lxxxi (1932), n. s. 34 (2), pp. 187-194.

[2]Metzer, H. J., and Hagan, W. A.: Corn. Vet., xvii (1927), pp. 35-44.

[3]Willman, J. P., Asdell, S. A., and Olafson, P.: Bul. 603, Corn. Univ. Agri. Exp. Sta. (1934).

[4]Hobmaier, M.: Arch. wiss. u. prokt. Tierh., lii (1925), pp. 38-47. *Ibid.*, liv (1926), pp. 213-222.

[5]Hjarre, A., and Lilleengen, K.: Virchow's Arch. 297, pp. 565-593 (abst. in Vet. Bul., viii (1938), 2, p. 113, Imperial Bureau of Animal Health, Weybridge, England).

[6]Madsen, L. L., cited by Maynard, L. A.: Animal Nutrition. (1st ed., 1937), p. 209 and 219.

[7]Bechdel, S. I., Honeywell, H. E., Dutcher, R. A., and Knutsen, M. H.: Jour. Biol. Chem., lxxx (1928), pp 231-238.

Hemorrhagic Septicemia in Indian Buffaloes

By TEODULO TOPACIO, B. S., D. V. M., M. S.,
Veterinary Research Division, Bureau of Animal Industry, Manila, P. I.

IN 1912, Mohler and Eichhorn reported the use of hemorrhagic septicemia vaccine in the control of an outbreak among the buffaloes in Yellowstone National Park. From all indications the epizoötic occurred sometime in 1911. In this country all the outbreaks of septicemia which have come to official notice occurred among cattle and carabaos. Since the introduction of the Indian buffaloes into this country in 1903,[1] as far as our records are concerned, the outbreak which is reported in this paper is the first of its kind to be studied in the field and diagnosed in the laboratory.

On June 14, 1938, a letter was received by Gregorio San Agustin, director of the Bureau of Animal Industry, from Dr. Pedro de Guia, superintendent of the Bongabon Stock Farm, Nueva Ecija, reporting the sudden death of a four-year-old grade Indian buffalo. This animal was reported missing shortly after the herd of 150 head was counted in the corral. On inspection of the pasture, the animal was found dead. The carcass was bloated and showed an advanced stage of decomposition, with the legs stretched, rigid and spread apart. Anthrax was suspected by Dr. Angel C. Dizon, farm veterinarian, and without opening the carcass, he collected blood smears from the jugular and ear vein, for microscopic examination. The result was negative and seven smears were forwarded to the Veterinary Research Laboratory for diagnosis. Again the result was negative. On the same day another ten-month-old bull calf was found moribund, and it finally died. Smears taken from the spleen, when examined microscopically, were negative. The remainder of the fresh smear, including two blood smears, were again forwarded to the laboratory for examination and diagnosis. Once more the result was negative.

From June 13 to June 20, seven animals died. The blood of the seventh animal, when examined in the laboratory, was again negative. These repeatedly negative results in laboratory examinations gave us the clue that septicemia was responsible for the malady. Hemorrhagic septicemia vaccine and anthrax vaccine were shipped to Bongabong with instructions to apply the hemorrhagic septicemia vaccine first, pending a final diagnosis. On June 20 all the buffaloes in the herd were given a single dose of hemorrhagic septicemia vaccine. With Drs. A. B. Coronel and Miguel Muñoz, the writer left for Bongabong to make a close study of the situation and to observe any recent carcass which might be found in the pasture. During two days of inspection in the buffalo herd pasture, only carcasses in an advanced stage of putrefaction were seen. One of these showed some fresh parts of the body, and smears were obtained from the inguinal vessels and precrural glands, which appeared to retain scarlet blood. When examined at the Provincial Hospital Laboratory at Cabanatuan (through the courtesy of Dr. Isip, director of the hospital), some Gram-negative bipolar organisms were observed in the smears. Although this evidence was by no means conclusive, it nevertheless prompted us to send a telegram to the Director of Animal Industry stating that "all indications are septicemia," pending autopsy of a fresh case.

Disposition of Carcasses

All carcasses found in the pasture by the joint party showed putrefaction of the same type described in the preceding paragraphs. In nearly all cases, the carcasses were found at the brim of the mud wallows—suggestive of the intense antemortem thirst of the sick animals. It was difficult if not impossible to distinguish the picture of the entire situation from a similar view in an anthrax outbreak. It is

in cases such as these that only the finding and isolation of the causative agent by laboratory methods will settle a dispute or a doubt in the field. The scouting party in a unit instituted field incineration of the carcasses, taking advantage of the liberal supply of firewood lying about the pasture. In every case this was accomplished on the spot. Not being able to find a fresh carcass for nearly 48 hours, we returned to the laboratory to await further developments.

FINAL DIAGNOSIS

On June 23, 1938, Dr. Ventura Gatchalian, District Veterinarian of Nueva Ecija and in charge of the vaccination work on the farm, reported the death of a ten-month-old calf 48 hours after vaccination. As the carcass was still warm when he found it, he collected fresh blood and a piece of spleen, packed the specimens on ice and sent them to our laboratory. These arrived at about 4:30 p. m. of the same day. Smears from the blood and spleen were stained and immediately examined microscopically. Again, all turned out negative.

A series of two rabbits, two guinea pigs, two rats and two mice were gathered. Each set of animals received a subcutaneous injection of blood and another subcutaneous injection of saline suspension of spleen at the rate of 5 cc., 5 cc., 1 cc., and 1 cc., respectively. In less than 17 hours all the animals died. Microscopic examinations of the heart blood revealed teeming numbers of Gram-negative bipolar organisms of hemorrhagic septicemia. The result was telegraphed immediately to Dr. Gatchalian and relayed to Dr. De Guia at the Bongabon Stock Farm in order that proper control measures might be taken accordingly.

GENERAL VACCINATION

In view of the proximity of the buffalo herd to the cattle herds and in order to avert a rapid extension of the outbreak, general vaccination with hemorrhagic septicemia vaccine, prepared by the method of the writer as described in 1933,[2] was ordered immediately after the diagnosis was established in the laboratory. The chute used in the vaccination is shown in figure 1. Specimens of a grade Indian buffalo steer and purebred buffalo bull are shown in figures 2 and 3.

Of 1,579 animals vaccinated in the farm, 150 were buffaloes, 1,432 were Nellore and grade cattle; 90 were work carabaos of tenants and private persons. Fourteen buffaloes in all had died before vaccination was begun, and only one buffalo calf ten months old died after vaccination. In this case it was presumed that the infection was already setting in when the vaccine was applied, since the animal died only 48 hours afterward.

In view of the extreme susceptibility of the buffaloes to Pasteurella infection, the herd was revaccinated five days after the first injection in order to insure protection. After the general vaccination of all the animals, no more cases followed and, up to the present writing, everything is quiet on the farm.

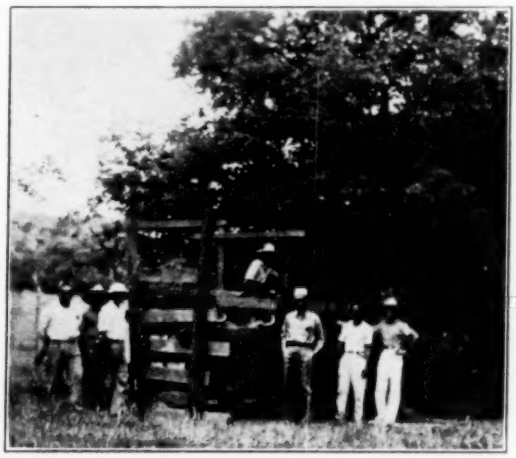


Fig. 1. Chute used in the vaccination of farm animals at Bongabon Stock Farm.

ISOLATION AND IDENTIFICATION OF THE ORGANISMS

As described in the foregoing paragraphs, considerable difficulty was encountered in the laboratory diagnosis of the smear specimens submitted from the field where successive examinations had proved negative. This difficulty is a common ex-

perience in some laboratories. In certain instances even the isolation of the organisms by cultural and animal inoculations has completely failed. A case in point is a report published in the Bureau of Animal

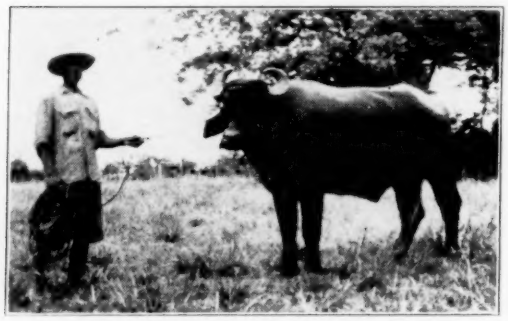


Fig. 2. A grade Indian buffalo steer.

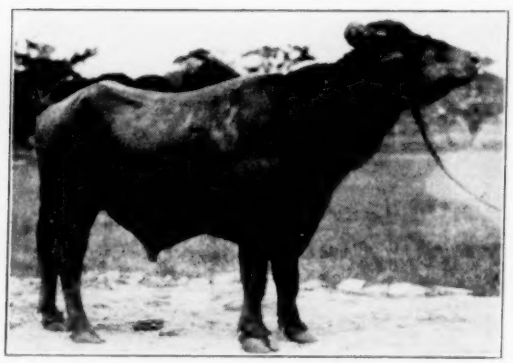


Fig. 3. Purebred Indian buffalo bull.

Industry Gazette in 1931.[3] In the present outbreak the last specimen from which the organism was isolated by direct animal inoculation of blood and spleen was negative by microscopic examination, and yet the rabbits, guinea pigs, rats and mice, which received separate injections of blood and spleen emulsion, died in less than 17 hours, showing large numbers of organisms in the blood. Cultures in broth and agar slants from these dead animals contained abundant typical growths of the *Pasteurella bipolaris bubalisepticus*. The organisms were nonmotile and Gram-negative, showing the characteristic bipolar staining.

Reaction in sugar media: In dextrose there was acid and no gas; in maltose there was acid and no gas; in inositol, no reaction; xylose, no change. This is characteristic of the group, although variations do occur.

Pathogenicity: Broth cultures 24 hours old killed rabbits in twelve hours when injected with 0.1 cc. subcutaneously. The point of injection was hemorrhagic and there were no visible lesions in the organs except congestions which characterize high virulence and toxicity.

ORIGIN OF INFECTION

Because the Cabanatuan-Bongabong highway runs through the middle of the stock farm and in spite of the complete fencing of the pastures, the origin of infection is difficult to determine. Under the circumstances the following may be considered as possible causes of the outbreak: (1) A sick carabao on the farm may have died, this death not being reported to the superintendent for investigation; (2) portions of infected carcasses might have been carried to the premises by dogs and other animals; (3) the abrupt changes from dry to rainy weather might have activated the organisms in the wallows or soil or in the animals themselves and thus caused the disease, and (4) other indirect sources of contamination from outside the farm might have been accountable. Although there was no outbreak of septicemia in the immediate vicinity at the time, it must be borne in mind that the disease is known to be enzoötic in Nueva Ecija.

SUMMARY

1. An outbreak of hemorrhagic septicemia in Indian buffaloes in this country is described and reported for the first time.

2. It has been shown that repeated microscopic examination of fixed blood or spleen smears from acute field cases of septicemia can not be entirely relied upon as a method of diagnosis in this disease.

3. Often even the isolation of the organisms from a carcass by cultural methods and animal inoculation fails.

4. Ordinary smears from the blood and spleen specimens, from which the organisms were isolated by animal inoculation, were entirely negative by microscopic examination.

5. General vaccination, accompanied by quarantine and disinfection of wallows, has been shown as an effective means of stopping the outbreak.

6. Of 1,589 animals vaccinated only one died, 48 hours after injection, which is suggestive of infection prior to vaccination.

ACKNOWLEDGMENT

For their coöperation in conducting the investigation of this outbreak, the writer is indebted to Drs. Pedro de Guia, Angel C. Dizon, A. B. Coronel, Miguel Muñoz and Ventura Gatchalian, and Dr. Elpidio Isip, director of the Provincial Hospital of Nueva Ecija.

References

[1]Report of the Philippine Commission, 1903, Part II, p. 664.
[2]Topacio, T.: A prophylactic vaccine against hemorrhagic septicemia (barbone). B. A. I. Gazette, iii (1933), 2, p. 111.
[3]Topacio, T.: An atypical case of hemorrhagic septicemia in the carabao. B. A. I. Gazette, i (1931), 10, p. 13.

Have You Heard This One?

There is a story going around about Ferdinand the Bull's mother. It seems that Ferdinand's mother became jealous of her offspring's popularity and finally inveigled her owner into sending her to America. She went straight to Hollywood, but the surroundings did not fit her aristocratic tastes. Therefore, she was placed in a boxcar and sent to Kentucky to feed on the state's famed blue grass. In two weeks she mood indigo.

On to Memphis

Next to arthritis, orchitis is the most frequent complication of undulant fever in man. It can be demonstrated experimentally in the guinea pig, and has been observed in all of the farm animals except the horse. In rams, boars and bulls, brucellic orchitis is capable of spreading the infection.—*Lafenetre and Roman.*

On to Memphis

A herd of over 9,000 caribou, passing in front of a Canada-bound train, delayed it for nearly a day.

About the Thyroid*

Thyroidectomy is well supported in adult animals. Only certain degrees of apathy and indifference are noticed, and these may be absent.

Secondary conditions such as exposure to extreme temperatures, may, however, cause depression in thyroidectomized adult animals.

The thyroid undergoes regression in hibernating animals and an injection of thyroxin interrupts the hibernal sleep.

Thyroidectomized females are generally sterile. If gestation survives, the young are less numerous, suffer from eczema, and become cachectic and die.

In thyroidectomized dogs and swine, whether the skin shows the presence of infiltration (myxedema) or not, one can distinguish a type with turgescence and an atrophic type which is always seen in thyroidectomized cats, goats and fowl.

A bitch thyroidectomized by Vassal gave birth to two pups that did not survive, and another cited by the same author delivered eight pups, all of which survived save one. However, during lactation she suffered from convulsions. Perhaps loss of the parathyroids was the cause.

The larvae of thyroidectomized batrachians constantly show hindrance of metamorphosis (Hoskins and Schulte), although growth is not entirely arrested. The tadpole remains in the larval stage, perhaps through the persistence of the thymus, and undergoes a certain amount of acromegalia resulting from compensatory development (hypertrophy) of the pituitary gland.

As a matter of fact, the thyroid acts more to prepare for life in animals than to realize it.

*Excerpts from Revue de Pathologie Comparée, May, 1937.

The Cultural and Clinical Significance in Bovine Mastitis of Nonhemolytic Streptococci That Ferment Aesculin

By RALPH B. LITTLE, V. M. D.

Department of Animal and Plant Pathology, The Rockefeller Institute for Medical Research, Princeton, N. J.

THE PURPOSE of this paper is to describe the cultural and clinical significance of group III streptococci and other organisms that ferment aesculin in a small herd from which all cows infected with group I mastitis streptococci (*Streptococcus agalactiae*) were eliminated.

In the simplified cultural classification of the more common varieties of streptococci that are capable of causing mastitis, Minett[1] included in group III those that ferment salicin, mannite, aesculin and inulin, reduce methylene blue milk, and hydrolyze sodium hippurate. He reported that these strains may be responsible for either acute or transient infection with or without a marked change in the character of the milk, and that they have frequently been found in the secretion from apparently normal udders. Diernhofer,[2] in 1932, described these streptococci and applied to them the name *S. uberis*.

It is apparent from the studies of Plastridge *et al*[3] that in their experience the mastitis produced by aesculin-fermenting streptococci was usually mild and of short duration. Ferguson[4] described the cultural characteristics of 25 strains of streptococci that split aesculin and stated that on the whole the infections were sporadic and not widespread. Hereafter in this paper, typical streptococci which split aesculin and inulin (not raffinose) and reduce methylene blue milk (1:5,000) will be referred to as group III streptococci.

HISTORY OF THE HERD

The herd concerned in the present study is privately owned, and consists of approximately 19 cows maintained for the production of milk for family consumption. The cows are kept in an exceptionally modern dairy barn and the stable routine is carefully supervised.

The first bacteriological examination of the fore milk, in 1936, showed that seven purebred Guernsey and Holstein cows were infected with hemolytic streptococci belonging to group I, while two cows were infected in a single quarter with the group III streptococcus. The former animals were immediately disposed of, while the latter were maintained with the normal cows and milked last. Since the elimination of the seven diseased cows, the herd has remained free from infections attributed to group I streptococci.

During a period of 31 months since the first examination of the milk, 15 first-calf heifers have been introduced into the herd; two of these have been disposed of, one on account of sterility, the other because of a lactating rudimentary teat. Four older cows have been eliminated because of low milk production or breeding trouble.

METHODS

The fore milk from each quarter of every cow was examined bateriologically once a month, and the laboratory procedures already reported[5] were carefully followed. After the samples had been collected at the farm, they were kept chilled and immediately plated in blood agar. The leukocyte films also were prepared at the farm, and the chlorine dilutions set up.

In determining the reaction of the culture in aesculin, the broth was prepared according to the method described by Diernhofer.[2] The cultures were grown in the broth for 24 to 48 hours and then a drop of a 1 per cent solution of ferric citrate was added. In a positive reaction the solution turned dark brown. In discussing this reaction, Edwards[6] says:

> Harrison and Vanderleck (1909) were able to recognize colonies of *Bacterium coli* in aesculin-iron citrate agar by their black

colour. This was due to the fact that the colon bacillus acted on aesculin with the production of glucose and aesculetin, which formed a black compound in the presence of iron.

As noted by Plastridge *et al*,[3] another characteristic of a positive reaction before addition of ferric citrate is the loss of the blue fluorescence present in negative reactions and uninoculated tubes.

In preparing the medium for the cultural classification, the usual methods were used. As an additional precaution, however, fermentation tests were checked in media to which carbohydrates were added aseptically without subsequent heating of the medium.

RESULTS

Morphology: The streptococci were Gram positive and formed chains of short to medium length. Generally, the cocci were small, round and delicate in appearance.

Growth in Blood-Agar Plate: Following incubation periods of 16 to 48 hours, the colonies appeared to be nonhemolytic. The surface colonies were generally either flat or slightly convex, moist and glistening; yet occasionally the surface growth was slightly opaque. The deep colonies usually were biconvex and produced varying amounts of green pigmentation in the surrounding blood agar. In certain strains the color was very marked and could be seen with the naked eye, whereas in others the pigmentation could be detected more easily under the lens. On prolonged incubation or at room temperatures the green color tended to fade,

Growth in Bouillon: In the original transfer from blood agar, either dextrose or sterile horse serum was added to the broth in order to enhance the growth. The streptococci grew well in the bottom of the tubes as a flocculent deposit with a slightly turbid supernatant fluid.

Fermentation Reactions: The six strains produced about the same reaction in dextrose, the pH varying between 4.7 and 4.9. All of the cultures failed to ferment raffinose; but acid was formed in lactose, saccharose, maltose, mannite, inulin, salicin, sorbitol, and trehalose. The characteristic reaction in aesculin was demonstrated after incubation for 24 to 48 hours.

Reaction in Bile: Two strains grew sparingly on the surface of 40 per cent bile, and all the cultures were insoluble in bile.

Methylene Blue Milk: The reduction in color with the six strains occurred with a 48-hour incubation period.

Sodium Hippurate: All cultures hydrolyzed sodium hippurate with very little variation in the final reactions.

OTHER ORGANISMS THAT REACT IN AESCULIN

When the milk is examined bateriologically in the epidemiological study of mastitis, one may encounter streptococci and micrococci, other than the group III types, which split aesculin. Moreover, these atypical forms are likewise responsible for either mild or rather severe forms of udder disease. Strains recovered from the right fore quarter of cow 7 and the left fore quarter of cow 8 (see protocols) illustrate this point.

A summary of the more important cultural characters of the group III strains and miscellaneous organisms that ferment aesculin is given in table I.

It will be noted in table I that the six cultures regarded as typical group III streptococci present similar cultural characters, while the strain from cow 7 fails to ferment lactose, saccharose, raffinose, and inulin, or to hydrolyze sodium hippurate. The culture from cow 8 fails to ferment inulin and sorbitol, but grows well in the presence of 40 per cent bile.

NOTES ON INDIVIDUAL COWS

Cow 1, a nine-year-old purebred Guernsey, calved the last time on May 23, 1937, and was sold in July, 1938, on account of sterility. Group III streptococci were detected in the right hind quarter seven months after the first examination of the fore milk, and thereafter were present in the secretion up to the time of disposal. The bacteria counts varied between 2,000 and 30,000 streptococci per cc. The infection resulted in a distinct induration of the quarter.

Cow 2, a nine-year-old purebred Holstein, calved for the last time July 2, 1938. Group III streptococci were recognized in the milk from the left fore quarter on the first examination (1936) and thereafter on the next nine tests. After the next parturition of June 29, 1937, they were not found in the milk from this quarter on 15 monthly examinations. The character of the secretion was always normal, yet the infection resulted in a slight induration of the quarter. In a monthly examination on December 14, 1938, infection was again apparent in this quarter, as shown by the number of leukocytes (522,280 per cc.), the increase in alkalinity, and the presence of streptococci. Either the original strain was dormant in the udder during a period of 21 months, or the quarter became reinfected with the same cultural type of streptococcus.

Cow 3, an eight-year-old purebred Guernsey, calved last on December 7, 1937. Group III streptococci were cultured in the milk from the right fore quarter on the first examination (1936) and thereafter for 14 months. Streptococci were not detected in the milk on the last eleven monthly examinations. The quarter was slightly indurated, but the secretion showed a normal pH, chloride content, and cell count.

Cow 4, a five-year-old grade Holstein, last calved September 11, 1938. A mild transitory infection of one-month duration was observed in the right fore quarter on the ninth monthly test. Fourteen examinations thereafter failed to reveal the presence of group III streptococci in the secretion.

Cow 5, a grade Holstein first-calf heifer, calved for the second time October 20, 1937. She was sold March 11, 1938, on account of low production and the presence of a rudimentary teat just above the left hind one. This made clean milking difficult, because of occasional seepage of milk. Group III streptococci were recovered from the milk of the left hind quarter on the first bacteriological examination, conducted four days after parturition. Subsequent monthly tests over a period of seven months revealed their presence in the milk on two occasions. Seven months after parturition, these streptococci appeared in the secretion of the left fore quarter and, up to the time she was dried off, they were observed on six monthly examinations. After the second calving, they were present in the left fore quarter on six out of seven examinations.

TABLE I—The cultural characters of group III streptococci and atypical organisms which react in aesculin.

Culture	Morphology	Dextrose	Lactose	Saccharose	Maltose	Mannite	Raffinose	Inulin	Salicin	Sorbitol	Trehalose	40 Per Cent Bile Agar	Aesculin	Methylene Blue Milk (1:5,000)	Sodium Hippurate
Group III streptococci. Strains from cows 1 to 6, inclusive	Small, round, short, chained streptococci, sometimes occurring in small, loose, irregular clumps	4.7 to 4.9	+	+	+	+	−	+	+	+	+	−*	+	+	+
Atypical strain from cow 7	Elongated cocci, usually in pairs, but often appearing in short chains	4.1	−	−	+	+	−	−	+	+	+	−	+	+	−
Atypical strain from cow 8†	Lance-shaped cocci, usually in pairs but often appearing in short chains	4.3	+	+	+	+	+	−	+	−	+	+	+	+	+

*Two cultures show a very slight growth.
†Strain from cow 8 possesses some of the characteristics of *Streptococcus lactis*.

and in the left hind quarter on two out of five. Induration developed in all the quarters but was more marked in the infected two.

Cow 6, a grade Holstein, calved for the first time on September 29, 1936. Monthly bacteriological examinations of the fore milk throughout two lactation periods at no time revealed the presence of streptococci in the secretion from any quarter. She was dried off August 27, 1938, without subsequent milking, by simply reducing the daily amount of food given. The cow calved for the third time November 7, 1938, with mastitis in the left hind quarter. The secretion was scanty, thick and off color, and the quarter was markedly atrophied. The laboratory examination of the milk on November 21 showed a high alkalinity, a high percentage of chlorine, and a leukocyte count above 13,000,000 cells per cc. The bacteria count was 6,720,000 per cc. and group III streptococci were present in pure culture.

The last negative laboratory examination prior to the calving of November 7 was that of August 8, so that it is possible that the infection occurred following this examination or during the dry period. It should be mentioned here that the sphincters of the teats were exceptionally patent, with seepage of milk after the udder had been cleansed before milking.

Cow 7, a grade Guernsey, calved the first time late in the fall of 1935. The milk was examined twice during the termination of this lactation, and monthly during the succeeding two. Following the last parturition on August 9, 1938, small numbers of aesculin-splitting streptococci were identified for the first time in the milk from the right fore quarter, and on the next monthly examination their numbers had increased. The infection, although mild, had apparently developed during the dry period.

Cow 8, a grade Holstein first-calf heifer, calved for the second time on January 20, 1938. Aesculin-positive, micrococci-like organisms were observed in the secretion from the left fore quarter on the first monthly test. At this time the leukocyte count was 4,066,260 cells per cc. On twelve subsequent monthly examinations, these organisms were identified on eight different tests; whereas on four examinations, if present in the milk, they were in such small numbers that culturally it was impossible to detect them in fore milk samples. The monthly leukocyte count on four examinations was above 1,000,000 cells per cc. Following the second parturition, these organisms have not been detected in the fore milk, middle milk, or strippings on nine monthly tests. Moreover, the other determinations, such as pH, percentage of chlorine, and leukocyte count, indicated normality. The infection, however, resulted in a slight atrophy and induration of the quarters without interfering, however, with the milk production.

DISCUSSION

Of the streptococci that possess the ability to ferment aesculin and inulin, the six strains studied are culturally similar to mastitis streptococci of group III, described by Minett.[1] In the bacteriological examination of samples of fore milk, however, aesculin-splitting streptococci are occasionally encountered that fail to show these group characters. Certain strains differ only in their inability to ferment inulin,[7] while others show a marked variation in their reactions in the common differential tests. The strain isolated from the right fore quarter of cow 7 offers an illustration of this difference.

Thus it is readily seen that the aesculin medium is useful in the identification of group III streptococci or of closely related types. Moreover, since mastitis streptococci belonging to groups I and II (Minett[1]) fail to attack aesculin, its use as a routine diagnostic method is furthermore valuable in the rapid differentiation of the mastitis streptococci.[6]

The results of this study indicate that usually group III streptococci are responsible for a benign infection in the udder with only a slight alteration of the secretion, but that occasionally they may produce acute cases of mastitis (cow 6). Although the majority of the infections in this herd were mild, with apparent recovery in nearly

half of the animals, atrophy and fibrosis of the udder did develop in certain quarters. In this herd infections due either to group III streptococci or to atypical types which react in aesculin were of considerable significance, since over a period of 31 months, eight of the cows were at some time infected, usually in a single quarter. Sporadic cases did occur, since four became infected, and a single quarter of two first-calf heifers was involved at parturition. Yet in most dairy herds the mild infections would have been entirely overlooked unless the fore milk had been tested monthly by as searching a bacteriological examination as was carried out in this herd.

It might appear that cows 2 and 3, which were originally left in the herd, were responsible for the gradual spread of the group III streptococcal infections. It so happens, however, that both of these animals recovered; and cow 2, a high producer (infection reappeared after an interval of 21 months), was kept in a box stall away from the other cows and milked separately. Furthermore, the two first-calf heifers had never been in close contact with the milking cows. In this herd, before milking, the udder of each cow was washed and dried with two separate cloths provided for each animal. These cloths were laundered daily. The attendants washed their hands in warm, soapy water before milking a cow. With all these precautions, it would seem that the chance of direct transmission at milking was very slight.

It is significant that during a period of 31 months after the first bacteriological examination of the fore milk, no new cases of group I infection have appeared in the herd, since usually this is the more common form of mastitis. Minett *et al*[8] have suggested that chronic mastitis due to group I streptococci is a contagious disease, and that such streptococci are obligatory parasites usually persisting only in the udder and its secretion. Their observations and those of Plastridge *et al*,[9] together with the information obtained in this herd, substantiate the suggestion first proposed by Minett and his coworkers that when cows infected with group I streptococci are removed from a herd, a reservoir for the organisms is eliminated, since the remaining cows can then be maintained free of infection. On the other hand, it may be impossible to develop a streptococcus-free herd until more is known concerning the source of group III streptococci and the varieties usually responsible for the more acute forms of mastitis.

SUMMARY

The results are given of observations on the cultural and clinical significance of group III streptococci in a small, self-contained herd of cows from which the animals infected with group I streptococci had been eliminated.

ACKNOWLEDGMENT

The author is greatly indebted to Drs. L. J. Tompkins, S. H. Johnson, G. H. Hopson, and F. W. Andrews for their coöperation in conducting the physical examination of the udders of the cows in the herd herein reported, and to Mr. Edward J. Foley for valuable technical assistance throughout this study.

References

[1]Minett, F. C.: Proc. 12th Internat. Vet. Cong., ii (1934), p. 511.

[2]Diernhofer, K.: Milcharstsch. Forsch., xiii (1932), p. 368.

[3]Plastridge, W. N., Anderson, E. O., Brigham, G. D., and Spaulding, E. H.: Conn. Agri. Exp. Sta. Bul. 195 (1934).

[4]Ferguson, J.: Cornell Vet., xxviii (1938), p. 211.

[5]Little, R. B.: Cornell Vet., xxviii (1938), p. 23.

[6]Edwards, S. J.: Jour. Comp. Path. & Therap., xlvi (1933), p. 211.

[7]Stableforth, A. W.: Jour. Path. & Bact., xlv (1937), p. 203.

[8]Minett, F. C., Stableforth, A. W., and Edwards, S. J.: Jour. Comp. Path. & Therap., xlvi (1933), p. 131.

[9]Plastridge, W. N., Anderson, E. O., Weirether, F. J., and Johnson, R. E.: Jour. Dairy Sci., xix (1936), p. 641.

Vicksburg, Mich., has passed an ordinance to regulate and control the keeping of cows within the corporate limits. One of its provisions requires that all owners of cows have them tested for tuberculosis and Bang's disease by a competent veterinarian.

Some Tests with Crystal-Violet Vaccine for Prevention of Hog Cholera

By B. H. EDGINGTON, D. V. M., and
A. F. SCHALK, D. V. M.
Animal Disease Laboratories, Ohio Agricultural Experiment Station, Reynoldsburg, Ohio

NUMEROUS attempts have been made to develop a satisfactory preventive or curative treatment for hog cholera. The work of Dorset, Bolton, and McBryde, leading to the discovery of the etiology of hog cholera, and the subsequent work of Dorset, McBryde and Nils, which led to the development of anti-hog cholera serum, represent the most noteworthy accomplishments in investigations of this disease.

The use of anti-hog cholera serum and virus for the immunization of swine represents one of the most successful utilizations of biologic therapy in farm animals. However, the use of virulent hog-cholera virus in the serum-virus simultaneous treatment entails an element of danger that limits the use of this method of prevention in attempts to eradicate the disease. The ideal agent for such a purpose should be relatively inexpensive, productive of a durable immunity, and be incapable of producing disease.

It is most fitting that one so eminent in scientific ability as the late Dr. Dorset should continue his research for an ideal immunizing agent against hog cholera, even though his earlier investigations had developed a product of inestimable value. The use of crystal violet, by Dr. Dorset and his associates in the U. S. Bureau of Animal Industry, as an attenuating agent for the virus, represents the most promising development in an extended series of attempts to produce a satisfactory vaccine against hog cholera.

Interest in this vaccine was stimulated through the release in December, 1935, by the U. S. Department of Agriculture of a "Tentative Outline of Production of Crystal-Violet Vaccine Against Hog Cholera," which apparently was intended to encourage production and tests of the vaccine. Shortly following publication of this outline, experiments were projected by the writers in an attempt to obtain additional data upon some phases relative to the use of the crystal-violet vaccine.

A progress report on crystal-violet vaccine for the prevention of hog cholera was published in December, 1936, by Drs. McBryde and Cole,[1] in which they presented the results of some of their work in the production and use of this vaccine. Several different combinations of crystal violet with other chemicals were used. The one most frequently tested and designated as "regular vaccine" was prepared by adding ten parts of a 1 per cent aqueous solution of phenol to nine parts of defibrinated blood obtained from pigs sick with cholera. Five parts of 1 per cent aqueous solution of crystal violet was then added to 100 parts of the previously phenolized blood. This mixture was incubated at 37.5° C. for 14 days.

Report was made of the results obtained from 271 pigs treated with crystal-violet vaccine. Slight reactions occurred in 9 per cent of the animals following injection with the vaccine. Adequate protection against inoculations of hog-cholera virus was reported in 98 per cent of the vaccine-treated pigs. A lapse of two to three weeks seemed to be required for the establishment of immunity following vaccine administration. The exact duration of immunity was not established, but in some tests it was found to be at least six months. No deterioration in antigenicity of the vaccine was noted when it was held in cold storage for as long as 14 months. The vaccine in either 5 or 10 cc. amounts was reported to protect pigs weighing 40 to 90 pounds against injections of hog-cholera virus. Subcutaneous administration was found to be superior to intraperitoneal injection.

Munce,[2] in 1937, reported results obtained with ten lots of vaccine used in a total of 120 pigs. The vaccine was prepared in a manner similar to the "regular vaccine" reported by McBryde and Cole. Immunity tests on 118 of these pigs were made 21 to 201 days following vaccine treatment, either by injecting hog-cholera virus into the vaccine-treated pigs or by their pen association with cholera-sick pigs. As a result of these exposures, 21 died, 17 showed severe reactions, five manifested mild reactions and 75 remained well. Irregularity in the degree of immunity was reported as the most unfavorable feature of the results obtained.

Several conditions arise in connection with what may be considered as the practical use of the vaccine. Some of the questions upon which information is desired are: (1) What is the duration of immunity following vaccine treatment? (2) Will hog cholera be transmitted by vaccine-treated pigs to cholera-susceptible pen mates? (3) Will equally satisfactory results follow the treatment of pigs from immunized sows and those of cholera-susceptible sows? (4) Will a satisfactory immunity be developed when the vaccine treatment is given during the phase of passive immunity which follows the administration of anti-hog cholera serum? A series of tests have been made to obtain information on some of the questions.

Preparation of the Vaccine

Fourteen different lots of vaccine were prepared and used in this series of tests. Each lot was prepared in the same manner and consisted of 90 parts defibrinated blood of cholera-sick pigs, to which were added 10 parts of 1 per cent aqueous phenol solution and 5 parts of 1 per cent aqueous solution of crystal violet. This mixture was put into hard-glass containers and incubated for 14 consecutive days at approximately 37.5° C., during which time the vaccine was gently shaken each day. After preparation the vaccines were kept under conditions of refrigeration until they were used.

The defibrinated blood used in the preparation of these vaccines was obtained from pigs that showed evidence of acute disease as a result of hog-cholera-virus inoculations six to eight days prior to their exsanguination. One vaccine, 2-c, was prepared with the blood of one pig. Three vaccines, 1-c, 2 and 14, were each produced with blood from 2 pigs. The other ten vaccines were each prepared with blood obtained from the pooled bleedings of 20 to 30 virus pigs. These latter lots of virus blood represented routine bleedings collected at a commercial serum laboratory under federal inspection.

The crystal violet used in the preparation of the vaccines was produced by Coleman and Bell Co., and was designated by them as lot 852, certified. Complete solution of the dye was assured by gentle heating, as suggested by Chapin.[3]

Animal inoculations and cultural tests were made to determine the presence or absence of contaminating microörganisms in the vaccines at the termination of their incubation. Vaccines 1-c, 2-c, 4-c, 5-c, 6-c, 7-c, 8-c, 9-c and 11-c showed bacterial contamination. *Salmonella suipestifer* only was isolated from vaccines 1-c, 2-c, 7-c and 10-c. Vaccine 6-c contained *S. suipestifer* associated with other organisms. The remaining bacterially contaminated vaccines contained microöganisms other than *S. suipestifer.*

Vaccine Treatment of Pigs

The pigs to which vaccines were administered, unless otherwise stated, were from sows that were not known to have been immunized to hog cholera. The litters of pigs in some instances were from different farms. The animals varied in weight from 20 to 100 pounds. All apparently were healthy at the time of treatment. The susceptibility of the pigs to hog cholera was ascertained by the injection of virulent hog-cholera virus into representative animals of each litter.

Some of the vaccines had been prepared for different lengths of time before they were used. Five or 10 cc. of the vaccines was injected subcutaneously into each pig. When 10 cc. of vaccine was used, the

amount was divided and injected at two different locations.

Slight temperature elevation was observed in a few of the pigs during the two weeks immediately following the vaccine treatment but, with one exception, none refused feed at any time and all rapidly returned to normal. These slight reactions were not restricted to pigs receiving bacterially contaminated vaccines.

MINOR TESTS

In the majority of instances, the vaccines were administered within a few days following completion of their incubation. This use of the vaccine primarily was intended to check their virulence. The immunity tests were made to determine whether or not the vaccines had antigenic value. With the exception of those of vaccine 14, these tests were made at relatively short intervals following administration of the vaccines.

The immunity of the vaccine-treated pigs was tested by injecting them with virulent hog-cholera virus. These virus injections were made at varying lengths of time following vaccine treatment of the pigs. None of the virus-injected pigs showed more than slight temperature elevations and these were of short duration. As a whole, the results of these tests were complementary to the crystal-violet vaccine.

SUPPLEMENTAL TESTS

The length of time that protective immunity may be retained by vaccine-treated pigs is a question of outstanding importance. A vaccine producing an immunity for less than six to nine months would be of little value in the usual scheme of swine production. Therefore it was deemed advisable to run additional experiments in which the immunity tests would be made at longer intervals following vaccine treatment of the pigs than in former tests.

Since the use of anti-hog cholera serum has been reported[1,2] as interfering with the production of immunity by crystal-violet vaccines, it was of interest to determine whether or not a degree of immunity might be presented in pigs from serum-virus treated sows that would have an adverse effect on the results of the vaccine treatment. This phase of the problem is of particular importance, as a large percentage of pigs in sections wherein the serum-virus method of immunization against hog cholera is a common practice are bred from immunized stock. Undoubtedly, pigs of such origin would be given vaccine treatment, should vaccine be used in such regions.

Information on these phases of the problem were sought through additional experiments using the bacterially sterile vaccine 2.

TESTS WITH VACCINE 2

Two groups of 40 pigs each, one from serum-virus treated sows, the other from nontreated sows, were used in the test. The groups were designated as immunized and nonimmunized sow's pigs, respectively. Some of the pigs from the immunized sows had just been weaned. The pigs of both groups appeared to be in normal health at the time of vaccine administration.

Twenty pigs from immunized sows and 30 pigs from nonimmunized sows were each given 5 cc. of the vaccine subcutaneously. The remaining pigs were held in their respective groups as controls on the possible spread of cholera infection either from the vaccine-treated pigs or other environmental conditions.

Each group was held as a separate unit in a well ventilated and bedded pen which had an adjacent outside enclosure. The two groups of pigs were separated by a wire fence and were cared for by the same attendant. Grain was fed twice daily in troughs situated in the outside lots and water was available to the pigs at all times.

REACTIONS TO VACCINE 2

During the 30-day period immediately following administration of the vaccine, the animals were under close observation. The temperature of each was recorded daily during the first three weeks.

Varying degrees of temperature elevation were noted. In some instances these elevations were definitely above what could be considered a normal range. The periods of elevated temperatures were usually inter-

mittent and seldom occurred for more than two to three days in succession. These temperature elevations were observed somewhat more commonly in the pigs from nonimmunized than in those from immunized sows. None of the pigs in the latter group refused feed, whereas several in the former group were off feed. This refusal of feed was usually for but one day, although occasionally it occurred for two days. One control pig and two vaccine-treated pigs in the group from nonimmunized sows died. All the remaining pigs apparently were normal 30 days after the vaccine had been administered.

The control pig died the 15th day following vaccine treatment of its pen mates. It had an elevated temperature for four days and refused feed for two days before death.

Postmortem examination revealed a large abscess in the inguinal region, apparently the result of castration. No lesions of hog cholera were observed.

One of the vaccine-treated pigs died on the 19th day following treatment. This animal showed temperature elevation for five days and refused feed for two days, prior to death.

Postmortem examination revealed rather extensive subserous hemorrhages of the stomach and bladder. Petechial hemorrhages were present in the kidney and beneath the epicardial and endocardial membranes. The intestinal mucosa contained a few petechial hemorrhages and was covered with a catarrhal exudate.

Heart blood collected from this pig at autopsy was injected into rabbits, white mice, pigeons and a supposedly cholera-susceptible pig. It was found later that an error had been made in the selection of this animal, since it had been injected with anti-hog cholera serum 44 days previously. This serum injection may have influenced the cholera susceptibility of the pig.

Neither the pigeons nor the pig showed reactions following the injections. The rabbits and mice died and *S. suipestifer* was recovered from each.

The other vaccine-treated pig died on the 22nd day following treatment. This pig showed very slight temperature elevations for one or two days interspersed with varying periods of normal temperature. Feed was refused for six days before death. The liver of this animal was pale and quite fibrotic. Numerous ascarids were present in the small intestine and were so densely packed at one place as to occlude the lumen of the bowel. Other lesions were not observed.

While these postmortem observations and animal inoculations do not afford unimpeachable evidence of an absence of hog-cholera virus in the pigs that died, nevertheless the presence of virus is challenged by the fact that none of the remaining 14 control pigs acquired the disease as a result of pen association for 39 days in their respective groups. Following the 39-day period of pen association, control pigs were removed from their groups and the vaccine-treated pigs were turned together on pasture.

The control pigs were then inoculated with virulent hog-cholera virus and died as a result of the infection.

IMMUNITY TESTS 3 MONTHS AFTER ADMINISTRATION OF VACCINE 2

Approximately 90 days following administration of the vaccine, eight pigs from the nonimmunized sows and ten from the immunized sows were each injected with 2 cc. of hog-cholera virus.

The fifth day after the virus injections, all of the pigs except two showed marked temperature elevations and the majority refused feed. During the succeeding twelve days, eight of these pigs died, one in a moribund condition was killed, two remained well, four had mild reactions and three showed severe reactions.

The sixth day after virus inoculation of the above pigs, another group of six vaccine-treated pigs were added to the pen. Five of these pigs were given serum-virus treatment immediately prior to their being placed in the pen. One received no additional treatment. All of the serum-virus treated pigs remained well throughout their contact with the others. The vaccine-treated pig that was given no additional serum treatment died on the eleventh day of its pen exposure.

The tenth day following the original inoculations of virus, another lot of five of the vaccine-treated pigs and three cholera-susceptible pigs were added to the pen containing the sick animals. Two of the vaccine-treated pigs became sick and died. The other three became ill but eventually recovered. The three cholera-susceptible pigs became sick and died.

The results of these immunity tests at approximately 90 days after vaccine administration are summarized in table 1.

The course of the disease; the temperature reactions of the virus-injected and pen-exposed vaccine-treated pigs; the postmortem findings of pigs that died; the development of cholera in susceptible pigs inoculated with filtered blood from sick vaccine-treated pigs; the development of cholera in susceptible pigs put in the pen with the sick vaccine-treated pigs; and the complete protection afforded vaccine-treated animals by serum-virus treatment administered immediately prior to their being added to the pen of sick pigs, all give evidence that hog-cholera virus was the primary cause of illness and death of these pigs.

However, such a deduction finds support when reference is made (table I) to the satisfactory results obtained in other immunity tests of this vaccine carried on 14, 21, and 40 days after vaccine administration.

The idea that a greater degree of immunity is required to protect against the injection of hog-cholera virus than is necessary to protect against pen contact with cholera-sick pigs appears to offer no explanation of the results following the exposure of the pigs in this test, since the vaccine-treated pigs exposed to hog cholera either by injection of the virus or through pen association with infected swine gave no evidence that they possessed a degree of resistance which was more protective against one than the other method of exposure.

The fact that the vaccine was approximately 50 days old when used in this test does not appear to be an explanation of the lack of immunity possessed by these pigs, since in other tests pigs treated with this vaccine when it was either younger or older showed satisfactory protection against hog-cholera-virus injections given at 14 to 40 days following vaccine treatment.

TABLE I—Results of immunity tests at approximately 90 days after administration of vaccine.

PIGS FROM	NO. TESTED	REACTIONS			
		NONE	MILD	SEVERE	DIED
Nonimmunized sows	9	1	2	1	5
Immunized sows	15	1	2	5	7

Twelve pigs in this test, exposed to the virus of hog cholera at approximately three months following their treatment with crystal-violet vaccine, died; ten exhibited varying degrees of illness but eventually recovered; and two remained well.

The conditions of this experiment do not permit an unqualified conclusion that the unsatisfactory results which followed virus exposure of the vaccine-treated pigs were due to a loss of immunity prior to the time of this virus exposure, since no immunity tests were made sooner than the 90th day after administration of the vaccine. However, such a deduction finds support when reference is made (table I) to the satisfactory results obtained in other immunity tests of this vaccine carried on 14, 21, and 40 days after vaccine administration.

The results of this experiment show no significant difference in the resistance to hog-cholera infection between vaccine-treated pigs from immunized and those from nonimmunized sows.

The lack of resistance in these pigs when exposed to hog-cholera infection 90 days after vaccine administration is at variance with the results reported by McBryde and Cole,[1] wherein the immunity of vaccine-treated pigs was of a satisfactory character when cholera exposures were made two to six months following vaccine administration.

The factor or factors responsible for this difference in results are not apparent. The possible influence of the origin and environmental surroundings of the pigs, a difference in antigenic value of the vaccine due to the nature of the crystal violet, or virus blood and crystal violet, used, and unintentional variations in methods of preparation and use of the vaccine, appear to be factors that might be involved.

In an attempt to study the influence of these factors in the production and use of the crystal-violet vaccine, parallel tests were proposed in which a comparison would be made of the results obtained with a vaccine furnished by the U. S. Department of Agriculture, Bureau of Animal Industry, and one prepared by the writers in accordance with the outline already referred to as having been released by the Department in December, 1935.

Immunity tests were to be made at approximately 40 to 90 day intervals following administration of the vaccines.

PARALLEL TEST OF VACCINES OHIO-14 AND U. S. B. A. I. "D"

Vaccine 14 was prepared in the same manner as the foregoing vaccines and was produced with blood obtained from two pigs six days following their injection with virulent hog-cholera virus.

Vaccine "D" was furnished by the U. S. Department of Agriculture, Bureau of Animal Industry, Ames, Iowa. The method of production of this vaccine was said to vary from that of vaccine 14, in that 10 per cent of a 3 per cent aqueous solution of anhydrous di-basic sodium phosphate was used instead of the phenol solution.

Different lots of crystal violet and virus blood of different sources were used in the preparation of each vaccine. The experimental animals were under identical environmental conditions. The 37 pigs used in this test represented six litters, five of which were from the same farm. Pigs used in this experiment were older than those used in former trials, to avoid any possible adverse effect on the antigenic action of the vaccine that might result from immunity present in recently weaned pigs.

Fourteen of the pigs were injected with vaccine 14 and an equal number received vaccine "D." Seven of the lighter-weight pigs of each group received 5 cc. and the remaining were given 10 cc. each of their respective vaccines. The pigs receiving 5 cc. of vaccine 14 averaged 84 pounds in weight and those given the same amount of vaccine "D" averaged 82 pounds. The animals receiving 10-cc. amounts of these vaccines averaged 107 and 106 pounds, respectively. Four pigs given no vaccine were held as pen controls. Five other untreated pigs used as controls on the susceptibility of the vaccine-treated animals were inoculated with hog-cholera virus and developed acute cholera. Evidence of illness was not observed in any of the pigs following administration of the vaccine.

IMMUNITY TESTS

Forty-two days after vaccine treatment, eight of the pigs were injected, each with 1 cc. of virulent hog-cholera virus. Four of these pigs had received vaccine 14 and four had been given vaccine "D." Two pigs of each group had received 5-cc. and two 10-cc. doses of their respective vaccines.

One pen-control pig was given a protective dose of anti-hog cholera serum while another received serum-virus simultaneous treatment. All of these pigs were under close observation for 15 days following the virus injections. During this time one pig that had received 10 cc. of vaccine "D" showed a slight reaction. The others remained well. All were apparently healthy at the termination of the observation period.

The original plan of the experiment called for virus exposure of another group of the pigs 90 days after the vaccine treatment.

Seventy-five days after vaccine administration, however, one of the remaining two pen-control pigs was slightly off feed. Temperatures of these pigs and the remaining 20 vaccine-treated pigs were taken. Both control pigs showed marked temperature elevations. All of the others were normal. During the succeeding six days, the vaccine-treated pigs showed no evidence of

illness, but the control pigs continued to have marked temperature elevations and refused feed. One of these pigs died on the 20th and the other on the 28th day after illness was first observed. Blood samples collected from these pigs on the fifth day of their illness were filtered through Seitz disks and the filtrates injected into three cholera-susceptible animals. One of these susceptible pigs received a dose of anti-hog cholera serum in addition to an injection of 5 cc. of each of the two filtrates. Each of the other susceptible pigs received 10 cc. of filtrate from a single source only. The pig receiving anti-hog cholera serum in addition to filtrates remained well following the injections. The pigs that received filtrates only became sick and died.

The eleventh day after the initial illness of the two pen-control pigs, four cholera-susceptible pigs were added to the pen. Two of these pigs were given anti-hog cholera serum just before they were added to the pen. The other two received no serum. Neither of the pigs given serum showed evidence of illness following their pen-exposure. The two pigs that received no serum became sick and died.

Beginning with the eighth day of illness of the pen-control pigs and for the succeeding two weeks, varying degrees of illness, as judged by temperature elevations and partial or complete loss of appetite, were observed in 14 of the vaccine-treated pigs.

The reactions manifested by these pigs are summarized in table 2.

The pigs showing severe reactions were ill only a few days. All had fully recovered within three weeks after onset of their illness.

To confirm further the opinion that hog-cholera exposure had been the cause of reactions in these pigs, each was injected with 2 cc. of virulent hog-cholera virus approximately 60 days after disease had been first observed in the group. It was thought, on the basis of former results, that had the infection to which these pigs were originally subjected not been hog cholera, at least some would show reactions following virus inoculations. If, however, their first reactions were the result of cholera infection, a similar recurrence would not be expected to follow virus injections. There was no evidence of health disturbance in any of these pigs following their inoculation with hog-cholera virus.

TABLE II—Reactions of the vaccine-treated pigs.

Number Pigs	Vaccine Given	Amount of Vaccine	Reactions			
			None	Mild	Severe	Died
5	No. 14	5 cc.	1	2	2	
5	D	5 cc.	2	1	2	
5	No. 14	10 cc.	2		3	
5	D	10 cc.	1	3	1	

The results of this test are somewhat more favorable to the crystal-violet vaccine than were those in the supplemental test of vaccine 2.

None of the pigs showed definitely unfavorable reactions in immunity tests made 42 days after the vaccine treatment. While none of the pigs died as a result of pen association with cholera-sick pigs approximately 75 days after vaccine treatment, there were several that had undesirably severe reactions. No significant difference was noted in the results of these immunity tests that could be attributed to an inherent factor of the vaccines used. If this be true in general, the cause of variation in results obtained by different workers must be sought in factors other than the vaccine used.

Our attention has been called[4] to the work of Hupbauer,[5] in which pigs supplied with sufficient Ca, P, and vitamins before they were immunized by serum-virus treatment, and likewise those which received these nutrients throughout the experiment,

withstood virus inoculations given 2½ to four months after immunization; whereas 5 out of 8 pigs supplied with an insufficient amount of Ca, P, and vitamins showed reactions following their injection with virus. If it be true that such dietary deficiencies influence the development of immunity to hog cholera, then the ration of the pigs in the parallel test of vaccines 14 and "D" merits consideration. As has been stated, the pigs in this test were maintained throughout the experiment indoors and in pens of concrete construction. Their ration consisted of shelled yellow corn and a protein supplement composed of equal parts by weight of cottonseed, linseed and soy bean oil meals and dry rendered tankage. While the Ca and P of this ration may be in a state of unbalance and the vitamin D content is unquestionably low, nevertheless none of the pigs showed the syndrome or gross lesions usually associated with rations deficient in these nutrients.

DISCUSSION AND SUMMARY

Within the limits of the tests conducted, no significant difference was observed which had relationship to the vaccines used, the history of serum-virus treatment of the sows from which pigs were obtained, or the period of time after weaning at which the pigs were vaccine treated.

The outstanding feature of these experiments is the difference in resistance to cholera manifested by animals in the immunity tests conducted 14 to 40 days and those made at 75 to 90 days, following vaccine administration.

A summary of the results of this phase of the tests show that, of the 36 pigs subjected to hog-cholera virus injections, 14 to 40 days following their vaccine treatment, none showed reactions of questionable severity. While in a group of 46 pigs subjected to cholera exposures 75 to 90 days following vaccine treatment, twelve gave undoubtedly severe reactions and twelve others died.

Pen association of 40 to 50 cholera-susceptible pigs with vaccine-treated pigs, for a period of approximately 30 days after vaccine administration, gave no evidence of spread of hog-cholera infection by the vaccine-treated pigs.

A comparison of results obtained through immunity tests of vaccine-treated pigs, the offspring of serum-virus treated sows and those from untreated sows, gave no evidence that a different degree of resistance had been produced in either group. Immunity tests of these pigs, made 90 days after they had been vaccine treated, gave evidence of an inadequate resistance to cholera, as shown by severe reactions and death of pigs. The degree of resistance appeared to be quite similar in either group.

A parallel test of vaccines produced at two different laboratories showed no significant difference in the antigenic properties of either vaccine. Immunity tests made by virus inoculation of representative groups of pigs, 42 days after administration of these vaccines, were highly satisfactory. Other pigs exposed to cholera by pen contact with infected pigs, approximately 75 days after the vaccine administration, showed a diminished degree of resistance.

What factor or factors may have been responsible for the unfavorable results obtained in immunity tests made 75 to 90 days following vaccine administration, other than those considered in the experiments herein reported, as yet remain undetermined.

It is recognized that results of tests conducted with so few animals as were involved in these experiments can not be accepted as conclusive. However, they do indicate the need of additional information regarding factors that may affect the successful use of crystal-violet vaccine for the prevention of hog cholera.

ACKNOWLEDGMENTS

The writers wish to express their appreciation of the aid given to this experiment by the U. S. Bureau of Animal Industry in supplying a vaccine, and especially for the constructive suggestions of Dr. C. N. McBryde.

We are also indebted to the Columbus Serum Company, Columbus, Ohio, for coöperation in supplying the virus blood used in the production of vaccines.

(Continued on next page)

Parasitic Skin Diseases*

By H. J. MILKS, D. V. S.
Cornell University, Ithaca, N. Y.

WHEN Dr. Zepp asked me to take a part in the discussion of skin diseases at this meeting, and asked whether I preferred to discuss parasitic or nonparasitic, I replied, "the parasitic," because I wanted to look into the subject myself. When I started to prepare this paper, I was somewhat undecided as to how to approach the problem, or, in other words, how I could present the subject in a different way. The diseases are well known and there is little new in their treatment.

I have been interested in them for approximately 30 years. My first paper on the subject was read before the Central New York Veterinary Society in 1914. This was in the era of creolin and other antiseptic baths for every sort of skin disorder. In that paper, I did not mention such baths except for fleas and lice, because I had never gotten very far with them, but had kept pretty close to information I had obtained from Hutyra and Marek, Friedberger and Frohner, Law, and others.

Fig. 1. Pustular type of follicular mange.

I was questioned about my failure to recommend the different antiseptic solutions, which at the time seemed rather unusual. That paper was published in the *American Veterinary Review* and the *Cornell Veterinarian* and, later, reviewed in one of the German periodicals, the reviewer commenting on my interesting way of handling skin diseases. This surprised me because most of my material and ideas had been taken from the German literature. It then occurred to me that people are all alike, and that a German did not have to be any more familiar with German literature than some of us Americans are with our own. I have given two papers on skin diseases before this section, and last year I discussed Dr. Dibbell's paper before the Eastern States meeting. Before proceeding any further, I want to disclaim any idea that I am a dermatologist and ask that some day I be permitted to discuss something else.

Why all this furor about skin diseases of small animals and dogs in particular? Formerly we almost never saw the subject mentioned on our programs and now no program or discussion is complete without it. The interest is certainly not attributable to our success in making discoveries, unless it is the discovery of our inability to handle or cope with them. I am afraid that with the nonparasitic group we must conclude that skin diseases are long drawn out or tend to be chronic, that their manifestations are on the surface of the body and, therefore, worry the owner more than internal conditions which of course he can not see. Restoration of function is not sufficient. The cure must be complete and the skin perfectly normal.

It is not clear why medicine as a whole

*Presented at the seventy-fifth annual meeting of the A.V.M.A., New York City, July 5-9, 1938.

(*Continued from page* 508)

References

[1]McBryde, C. N., and Cole, C. G.: Crystal-violet vaccine for the prevention of hog cholera. Jour. A. V. M. A., lxxxix (1936), n. s. 42 (6) pp. 652-663.

[2]Munce, W. T.: Experiments with crystal-violet hog-cholera vaccine. Jour. A. V. M. A., xc (1937), n. s. 43 (3), pp. 307-319.

[3]Chapin, R. M.: Personal communication.

[4]McBryde, C. N.: Personal communication.

[5]Hupbauer, A.: Zur frage des immunitatsdrchbruches nach simultanimpfungen gegen schweineseuche. Arch. wiss. prakt. Tierrheilk., lxxi (1937), 5, pp. 383-390.

has not found out more about this organ, the skin. It is external; we can see it, examine it with a hand lens, feel it and handle it, and yet have the impression that we know less about it than we do about the kidneys, most of the internal organs, or the mucosa of the gastrointestinal tract. Just think this over.

Parasitic skin diseases usually are divided into those caused by animal parasites and those caused by vegetable parasites or fungi. Among the former are follicular and sarcoptic mange, ear mange, lice and fleas. Ringworm, favus, and probably several other conditions comprise those caused by fungi. I am not going to give you any description of these parasites because you are already familiar with them.

FOLLICULAR MANGE

Follicular or demodectic mange is caused by the *Demodex folliculorum*. This parasite is quite closely limited to dogs but occassionally has been found in the horse, ferret and hog, and sometimes in the blackheads or comedones of man, where it is generally thought to be nonpathogenic. It was first described by Henle and Seman in 1841-42, who found it in the blackheads of man. It is said to be rare in children, but present in from 64 to 80 per cent of adults.

Symptoms: Follicular mange exists in two forms, the pustular and the squamous. In our experience the squamous has been much more frequent. The mite usually prefers the thin, tender-skinned portions of the body—inside of forelegs, lower side of neck, lower abdomen, inside of thighs, the face, especially the parts above the eyes, around the ears and the commissures of the lips.

The pustular type is shown by separate pustules, generally not larger than a pea, upon a skin described as copper red in color, although this does not always hold true. The skin is often thickened considerably and thrown into folds (fig. 1). The lesions start as papules which soon change into pustules. The pustules may coalesce, thus making a larger one. The contents of the pustules vary from a yellow to a sanguinous pus which, if examined under the low power of the microscope, will be found to contain numerous mange mites. The mites are located deep in the sebaceous gland and hair follicules. The pustules either break or are scratched open, making just the sort of mess that one would expect. New pustules form, the disease spreads and, unless checked, becomes generalized. In generalized cases the animal becomes emaciated and a very serious condition results.

The squamous form is entirely different. It also attacks the thin-skinned parts mentioned under the pustular form, to which must be added the skin around the ears and forehead above the eyes and around the commissures of the lips. The skin is ordinarily described in the texts as copper red, but this is not a good sign. More often it appears in excellent condition, except for the loss of hair. It is not thickened, is normal in color, just simply bald. This form frequently starts around the eyes, lips, etc., as mentioned previously, and sometimes as irregular bald spots scattered over the animal, much resembling certain forms of ringworm but not spreading so rapidly and the spots not being so regular in shape. See figure 2.

It is these localized, benign-appearing areas that are the starting point of red mange. The disease must start in some place and must be localized before it can become generalized. The prognosis is much better in these localized forms than in the generalized ones. Whenever you find the Demodex you have red mange. The localized cases, often picked up with the microscope and always confirmed with it, gave us a good percentage of cures before the days of rotenone. Our records show a 64 per cent record of recoveries from 1920 to 1931, when Crane described his results with rotenone.

Contagiousness: The contagiousness of demodectic mange has been discussed pro and con. It is not easy to transfer the disease experimentally, yet we know that there are kennels in which the crop of puppies are usually affected. We also know of many instances in which whole litters of puppies of bitches which had recovered from red mange became affected. It

is not clear just how this happens. Undoubtedly there are carriers whose skin remains sound. There also may be some inherent immunity that is hard to explain, conditions in which animals are peculiarly susceptible and in which the disease spreads very rapidly, such as following distemper or some other debilitating disease.

Diagnosis: A provisional diagnosis of demodectic mange may be made from the location and the appearance of the lesion, but this must be confirmed with the microscope, not so much because one needs it in typical cases as because this instrument will prevent one from going astray in the atypical, benign-appearing lesions. Most of us who have not used the microscope very much would never consider these lesions as red mange but later wonder why that insignificant lesion persisted and perhaps grew worse in spite of treatment. We have scraped hundreds of cases of suspected follicular mange and other skin disease and have encountered the Demodex mite so frequently in so many different appearing conditions that we are no longer sure of anything until we have examined a scraping. Consequently, we scrape most skin cases as a routine measure and our results sometimes surprise us.

Microscopic examination: In the pustular form one may simply squeeze out a drop of pus onto a glass slide and examine it in fresh condition under low power, with or without a cover, or one may add a drop of caustic solution or oil to the pus. We rarely use a cover for routine work. If we really desire to study the parasite, we use the cover. An important point is to keep the preparation wet; otherwise, things will be so distorted that nothing can be distinguished. Do not permit too much light through your condensor. Low power requires comparatively little light. Excessive light renders the mites too transparent to be seen. Close the iris diaphragm until the lighting is correct. For the squamous type, scrape deeply until blood or serum oozes. These parasites are located deep and it is necessary to go after them. A few scales rarely do.

There are two ways to handle the scrapings—caustic or oil. With the former, the material is placed in 10 to 20 per cent caustic soda or potash and allowed to macerate and so liberate the parasites from the crusts and scabs. Or, the process may be speeded up by heating. For many years we placed the scrapings upon the slide, added a few drops of caustic and boiled them by holding over a match.

Lately we have been using what we term

Fig. 2. Follicular mange, squamous type.

the oil method. This did not originate with us and we do not know of the one with whom it did. Essentially, in this method, oil is used as the clarifying agent. We either place a drop of oil (mineral or light machine) on the side of the scalpel to which we will scrape or a drop of oil on the part to be scraped. I believe all this does is to make the material stick to the knife. Then we transfer this mixture to a slide, add another drop or two of oil, stir a bit, and examine as described above. With this method the parasites are found alive. The method is very simple and may be used in searching for any variety of mite.

Prognosis: This has changed for the better since Crane's work with rotenone in 1930-31. Previous to that, we had an average of 62 per cent of recoveries of all cases seen in the clinic between 1917 and 1931, but that does not mean 62 per cent of generalized cases. I believe the microscope helped this percentage of recoveries because we had many early or beginning cases. Since we have been using rotenone, the percentage has jumped to 87 per cent,

including all varieties of cases. The remaining 13 per cent were not all incurable or hopeless. Several were not worth the treatment and were destroyed.

Treatment: Red mange is such a persistent disease that, previous to the use of rotenone, it seemed that everything had been used in its treatment—balsam of Peru, styrax, balsam of Peru and creolin, 10 per cent of each in alcohol, balsam of Peru in ointment, sulfur ointments, sulfur dips,

Fig. 3. Sarcoptic mange (foot rot) in a ferret.

kerosene, lime sulfur, etc.—with but poor results in old cases. For local lesions, we used tincture of iodine, balsam of Peru in ointment or alcoholic solution, and the agents mentioned previously. About 1915, we stumbled on the use of nitrate of mercury ointment (citrine ointment) which at that time we were using for ringworm. It appeared to work so well that we used it more and more in the generalized cases, as well as in the local ones, until rotenone appeared.

This really worked fairly well, but required a long time. Our preparation was citrine ointment 1, lard 3, which we later changed to citrine ointment 1, and neat's foot oil (genuine) 3, because this was easier to apply and seemed more effective. For well known reasons we still use this preparation around the eyes. Rotenone has so changed the treatment that it is rather easy to describe. There are so many formulae on the market that it appears that we are not all agreed as to which is the best. We still use much of the acetone alcohol mixture, as first described by Crane, but we have increased the acetone content to 20 per cent. There is some objection to the solution because it is rather irritating to some dogs and a little of the rotenone settles out, but it is cleanly and really is the solution upon which the early work was done. We know that this is dangerous about the eyes and, here, we use either 1 per cent in ointment or the citrine ointment, which we have never discarded. Canex and numerous other preparations on the market do so well that their discussion is superfluous. Our experience with various biological products, including bacterins, foreign proteins and organ extracts, has not been very encouraging, but no doubt these preparations have their place.

SARCOPTIC MANGE

Sarcoptic mange (the itch) is common in dogs and cats. We have also seen a few cases in the so-called "foot rot" in ferrets (fig. 3). Our experience with fur-bearing animals is almost nil and we know nothing about their skin diseases. Sarcoptic mange is a true scabies caused by the *Sarcoptes scabei var., S. canis* and *minor* (*Notoedres cati*). It is but occasionally transmitted to the human and other animals and back and forth between the dog and cat. It is readily transmissible from one dog or cat to another.

Symptoms: As in the case of the demodectic mange mite, the sarcoptic prefers the thin-skinned parts of the body. In dogs the choice appears to be around the head and ears, underside of the neck, in the axilla, lower abdomen, the inside of the front and rear legs and the inside of the thighs. It is spread readily from one part of the dog to another by the animal's scratching in this manner, furnishing free transportation for the "hitch hiker" to a new country. The early lesions are described as papules, vesicles, or pustules or the pin-point hemorrhagic areas in which the mite is supposed to be. Sometimes the burrows of the mite may be observed in the skin. The dog scratches itself incessantly,

particularly if placed in the sun or brought into a warm place, the scratched areas soon being transformed into open sores covered with crusts and scabs. The itching and scratching worry the animal, and he may lose weight and die of cachexia.

Diagnosis: This must be made from the location of the lesions and extreme itchiness way out of proportion to the lesion. The history may be the deciding point. "Where was the dog obtained?" is enough to clinch the diagnosis in some cases. "Have you other dogs and are they beginning to scratch?" is another leading question. If your nerve is pretty good, you may ask if any member of the family is affected with a skin disease which started subsequent to the disease in the dog. We ask this question frequently and have never angered any one yet. Finally, a microscopic examination, made exactly as described for follicular mange, is useful.

I prefer, in this disease, to make a composite scraping from several areas in sarcoptic mange. The sarcoptic mite is not so easily found as the Demodex, and therefore a negative examination would not be considered as conclusive or mean much more than our inability to find the mite. We can not help believing that it is foolish to discard the history, symptoms, and lesions just because we can not find the parasite with the microscope. Contrary to this, we frequently make a positive diagnosis in the face of a negative microscopic examination. One should think of the comparatively small amount of territory involved in making our scrapings and feel that time and persistence might reveal the mite or egg. Some of these cases are as evident as mange in the larger animals, in which it is sometimes facetiously said that one does not need a microscope, that he should be able to diagnose the disease with a telescope.

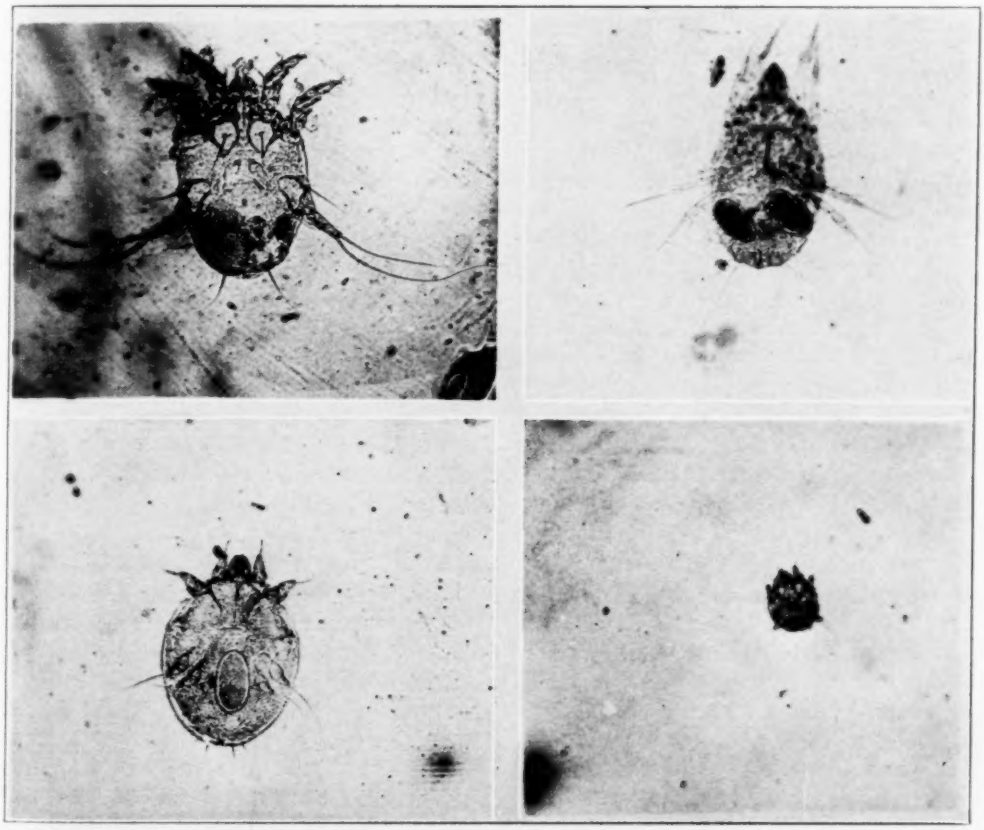


Fig. 4. **Upper left, ear-mange mite,** Otodectis cynotis; **lower left, sacoptic mite,** Sarcoptes carris; **upper right, feed mite,** Glycyphagus domesticus; **lower right, sarcoptic mite (cat),** Notoedres cati.

Occasionally the mites, both follicular and sarcoptic, may be found in the feces, but there is another insect, the grain mite, often present in large numbers in the dry granular feeds, that appears very similar to the sarcoptic mite and may be mistaken for it (fig. 4). This must be kept in mind because these mites pass through the animal in as good shape as do the mange mites and may thus cause confusion. They are often found in large numbers in other stock foods and there is little difficulty in finding them in the feces of animals which consume infested food.

Prognosis: This is good in dogs that are in good physical condition. The disease may be cured in the neighborhood of two weeks or less if carefully treated. In cats the prognosis is not so good, particularly if the animals are confined during the period of treatment. For some reason, many of these animals appear to get along pretty well with the mange until treatment is started, and then they simply drop off.

Treatment: Unlike the follicular mite, the Sarcoptes does not penetrate the hair follicules and sebaceous glands and is relatively easy to reach and to kill. There are some one-day treatments for human itch, but we have found it much safer to use a few continuous treatments to get the last parasites hatched before they have matured sufficiently to lay their eggs. Some form of sulfur or tar has been used for generations and is still the best treatment. Sulfur ointment, lime sulfur dip, potassium sulfide dip, or ointments containing tar, lysol, creolin, balsam of Peru, or betanaphthol may be substituted. Danish itch ointment depends largely upon potassium sulfide for its action. This agent, also known as liver of sulfur, is an old remedy.

A comparatively new remedy for itch in man is pyrethrin ointment N. N. R. Instead of using the ointment, we have used a preparation in oil of the same strength, pyrethrin in cotton seed oil. This is supposed to be a 24-hour cure in man. We have not trusted it that far, but its use appears promising. Many people are sensitive to pyrethrum and we have been a little afraid of such a condition in dogs, although we have not encountered it.

Another suggestion is to manufacture the hydrogen sulfide upon the animal instead of applying it in solution. This is done by thoroughly wetting the animal with a 40 per cent solution of sodium thiosulfate followed after 4 to 15 minutes with the application of 4 per cent of hydrochloric acid. We tried it once. The sulfide was liberated all right but the animal was found dead after a few minutes, probably from inhaling too much of the gas. One-fourth of 1 per cent of this gas is dangerous and 1 per cent may be fatal. The treatment might be satisfactory in a large, well-ventilated room.

In general, we believe that the thoroughness of the treatment is the secret of success. Topical application courts disaster, because there will ordinarily be a new area appearing at just about the time the old one is getting well, and we simply follow the disease from one portion to another, always running a poor second to the mite.

Everything considered, we believe that dipping of the entire animal is the quickest, easiest and best way to handle the disease. A dip twice a week for a couple of weeks should ordinarily be sufficient. There is just a word of caution about the home-made dips of lye and sulfur. Recently we had a chemist go over these and he found that not one is correct chemically. When we use lye and sulfur, we make either a sodium sulfide or potassium sulfide solution, depending upon whether we use sodium or potassium hydrate. At any rate, the result is a definite chemical compound and not a mixture.

Most of these dips do not contain enough lye to react completely with the sulfur, and the acid which is generally added to overcome the alkalinity is not only unnecessary but liberates the hydrogen sulfide which we want. It is much better to rely on a dip made from a definite chemical compound, such as lime and sulfur, either liquid or powder, or a solution of liver of sulfur.

In the cat the disease is usually found upon the face, ears, neck and, occasionally, other parts. The disease is so simple that it requires no further description. The microscopic diagnosis is usually much easier, and in this way many eggs of the parasite are frequently seen. The treat-

ment is about the same as for the dog, but the risk is greater. One of the dips is usually preferred to the ointments.

EAR MANGE

Ear mange is common to the dog and cat, rabbit, fox, and perhaps other carnivora. Some animals appear predisposed to it and others quite resistant. The disease manifests itself by extreme itchiness, causing the dog to shake the head violently or scratch the ears with the foot. Many torn ears result from the animal's wounding the ears on the collar or the other object during one of these shaking spells. When we see a dog with the edges of its ears torn, and in which the wounds will not heal, we always suspect parasitic otorrhea. If neglected, these cases may terminate in bad cases of catarrhal otorrhea, which are difficult to handle.

Some authors believe that parasitic otorrhea may be one of the causes of hysteria in dogs, and we are inclined to support that idea to some extent. It is a frequent cause of convulsions in cats; otherwise, the symptoms in these animals are just about what we should suspect—scratching of head behind the ears, and shaking of head to ease the itching. We have seen a few cats which have had severe wounds back of each ear caused by incessant scratching. Diagnosis is made from the symptoms and brick-red or brown color of the exudate. The ears are not tender in uncomplicated cases. Instead of resisting, the animal may crowd against the applicator with which the ears are being cleaned and make motions with the foot as if he were doing the scratching. The mites are large and sometimes may be seen as white specks upon the dark background of the exudate. Those of the rabbit can be seen readily on paper. The disease is much more easily diagnosed microscopically in the cat than in the dog, but we believe, as in sarcoptic mange, that we should not throw away our clinical knowledge just because we fail to demonstrate the mites in each case.

Treatment: Any of the agents recommended for sarcoptic mange and which are not irritants may be used. The ears should be first cleaned with oil or an alcoholic preparation before applying the medicine, and this should cover the area around the base of the ear and inside of the flap in order to catch any mites in these places. Recently it has been reported that the mites may be found on the tip of cats' tails. We have not noticed this. It is a point to keep in mind and may account for some of the relapses. The mites are not difficult to kill and yet the disease may be persistent, making it easier to control than to cure. We have noticed a resistance in some animals and a predisposition in others and are not certain whether the relapses which occur are really relapses or the result of reinfestation. The most common parasiticides used are oil of cade 1, cottonseed oil 8, mercuric nitrate ointment 1, neat's foot oil 3, phenol in glycerin, etc. The disease is evidently not so easily handled in dogs and cats as is reported in foxes.

Fig. 5. Ringworm in a Boston terrier.

RINGWORM

Ringworm is a term used to designate a skin disease caused by a fungus. In veterinary medicine, we think of tinea tonsurans as the cause of ringworm and tinea favosa of favus, the two best known and most common fungi found on animals. Veterinarians have known about these fungi for many years and little has been added to our knowledge in generations. Law in the *Farmers' Veterinary Adviser* (1874), lists the above two and two others, and states that chloroform bleaches ringworm hair white without affecting the sound hair.

Sutton's *Dermatology*, eighth edition, lists 19 conditions as due to fungi and devotes 98 pages to them. There is little doubt but that some day we shall associate fungi with some of the skin diseases that we do not understand at present. Microscopic diagnosis requires good equipment, good technic, and considerable experience. Differential diagnosis is made upon the cultural characteristics of the various fungi. Veterinarians as a group are not familiar with mycology and have to be content with finding a fungus. We are told that the subject is difficult.

Ringworm, herpes, tinea tonsurans,

Fig. 6. Favus in the dog.

herpes tonsurans, tinea trichophyton, are the forms ordinarily seen and meant when we speak of ringworm. It appears in at least two forms; first, the typical ringworm as seen in man, which spreads in irregular, enlarging circles with more or less sound tissue growing within the circle and second, the well defined, quite regular, bald spots which spread from the center, but with the margins uniformly sharp (fig. 5). Whether these two forms are due to the same fungus is a question for the mycologists to decide. The areas are not extremely itchy.

Diagnosis: This must be made from the characteristic spreading lesions in which typical cases could hardly be overlooked. Wood's filter upon an ultraviolet light is often of much benefit because many fungi fluoresce under this light. The filter simply removes the visible light rays. We believe that the fluorescence is positive of fungi, but some fungi do not fluoresce. Whether all pathological skin fungi do is the question, but it appears that there must be exceptions. A microscopic examination may reveal the spores and mycelia. This is made by digesting the scales or hair in strong alkali and examination under high power. It has been claimed for many years that chloroform would bleach the diseased hairs white. We are not prepared to discuss this subject.

Favus (fig. 6) or honeycomb ringworm, occurs as characteristic thick scales, more or less circular in shape with depressed centers caused by *Anchorion schonleinii*. Favorite parts attacked are paws, scalp, and occasionally other parts. Cats are believed to become infected from mice.

Treatment for ringworm, whether of one type or another, is in many cases practically the same, but a cure requires a long time—two to three months. Tincture of iodine sometimes gives good results as do strong alcoholic solutions 1 to 500 mercuric chloride; salicylic acid in solution or ointment and in combination with benzoic acid, Whitfield's ointment, is one of the standard treatments. Mercury nitrate, in ointment or oily solution, has been satisfactory in our hands. Ammoniated mercury ointment is highly recommended. Before treatment, the hair should be removed by extraction, depilatory, or shaving, and the crusts scrubbed off.

We believe that ringworm in cats is sometimes diagnosed as eczema. The disease here occurs in more diffuse, well scattered, small areas. These cases do not give typical fluorescence with Wood's filter, but do show rather bright blue instead. Occasionally we have found the spores and mycelia in these cases. In human ringworm of the scalp, an epilatory dose of the x-rays is given, or just the correct dose of thallium acetate to make the hair fall out. In this case, the hair and not the skin is diseased and, by the time a new crop is grown, the ringworm is gone. If too little x-ray is given, the hair does not fall out and there is no benefit; if too much, the hair does not return. Thallium acetate is even worse; too little does no good and too much is distinctly poisonous.

Some Nutritional Studies in Swine*

By H. E. BIESTER, V. M. D., and
D. F. EVELETH, D. V. M.
Veterinary Research Institute, Iowa State College, Ames, Iowa

THE INTERPRETATION or significance of demyelination of the myelinated fibers of the spinal cord is an extremely important point in the pathology of nutritional and infectious diseases. Some workers have attributed the change to a deficiency of vitamin A, while others have associated it with a vitamin B_1 deficiency. It has also been described as a part of the pathology of hog cholera. This degenerative change has also been associated with one form of so-called posterior paralysis of swine. During the course of investigations in this field, we have observed severe degenerations of the myelin sheaths in the absence of incoördination. In one experiment, field cases fed substances high in vitamin A recovered from the incoördination following treatment and regained the ability to walk and run. These clinically recovered cases presented spinal cord demyelination equally as severe as that of the controls that died.

In investigations on the effect of fluorine on dogs fed minute quantities such as are encountered in some water supplies, we observed myelin sheath degenerations in the spinal cord and fatty degeneration of the distal part of the spiral tubules of the kidneys of these dogs and also the controls to whose ration no additional fluorine was added. The animals were fed a modified Mellanby ration composed of the following:

180 cc. of fresh whole milk plus the following mixture (by weight):
30 ground yellow corn
30 ground hulled oats
23 Ideal dog food
10 skim milk powder
5 wheat germ
1 cod liver oil
1 NaCl containing 0.0,001 pt. NaI

The corn and oats were moistened with distilled water and autoclaved for 1½ hours at a pressure of 15 pounds. This ration contained ample quantities of vitamins A, B, D, E and G. While it will maintain dogs cheaply and satisfactorily, two criticisms might be directed against it from the standpoint of completeness. It is low in vitamin C and much of the protein is derived from cereals, which we know are deficient in some of the amino-acids, notably lysine and tryptophane.

Unpublished studies on several large series of rats have definitely shown that when large amounts of skim milk powder, rich in lysine and tryptophane, are included in yellow corn and skim milk powder rations, the renal degenerations fail to appear, but the spinal cord degenerations are not prevented.

The ration that is usually fed in attempts to produce incoördination in swine consists of 20 per cent tankage, minerals and white corn or kaffir. It is a known fact that tankage has a low vitamin content. Furthermore, the proteins and carbohydrates have been altered from a nutritional standpoint by the exposure to high heat during processing. The deleterious action of heat is illustrated when skim milk powder is autoclaved in order to destroy its B_1 content. When vitamin B_1 in the form of yeast is added to replace that destroyed in the heated skim milk powder, the heat-treated skim milk powder plus vitamin B_1 is not so efficacious nutritionally as an equal amount of untreated skim milk powder. It was for this reason that we substituted skim milk powder for tankage in subsequent experimentation in swine.

In one experiment, when brood sows or gilts were put on a ration consisting of 78 per cent white corn, 20 per cent skim milk powder and 2 per cent minerals and salts several months before breeding, a definite incoördination developed in the pigs. The picture also included scoliosis. The pigs ate more sparingly of the ration as time passed. Assay of the skim milk powder showed it to be very low in vitamin A. In

*Presented at the seventy-fourth annual meeting of the A. V. M. A., Omaha, Neb., August 16-20, 1937.

order to stimulate the feed consumption, skim milk powder was fed *ad libitum*. The affected pigs made rapid improvement and rapid weight gains.

An analysis of our previous researches led us to the hypothesis that the rations used in the production of posterior incoördination were deficient, not only in vitamin A, but that they lacked some of the essential amino-acids in which corn is deficient.

The following experiment was planned to include rations that contained adequate amounts of vitamins A, B, D, E and G. Vitamin C was injected as ascorbic acid (cebione) into two animals of each group. The influence of varying levels of protein, as well as its qualitative properties, is considered.

Table I shows the four experimental rations under consideration. The total Ca and P approach the average requirement of .700 elemental Ca and .300 P per 100 pounds in rations 1, 2 and 3. In ration 4, the total CA and P is practically twice the average requirement, although the Ca/P is similar to the others. These rations should contain ample vitamins A, B, E and G, as determined by tables of analyses and by our own assays using baby chicks. Two pigs in each lot received weekly injections of vitamin C in the form of ascorbic acid (cebione). The injections commenced with ⅛-gram doses weekly. This amount was gradually increased to 1 gram per pig per week.

The sows and pigs were fed these rations soon after farrowing. From June 2 to October 2, a period of 122 days, the gains per pig for rations 1, 2, 3 and 4 were 18.5, 25.5, 33.7 and 150.0 pounds, respectively. After being on the rations about four months, one pig from each group was destroyed for study. None of those sacrificed at this time had received vitamin C injections. Demyelination in the spinal cord was quite pronounced in the four pigs (table II). The variations in the protein and the increased total CA and P of ration 4 seemed to have no influence on this retrograde change. A fatty degeneration of the renal epithelium, confined chiefly to the medullary ray portion, apparently was correlated with the protein. In the pig from lot 4, receiving 50 per cent skim milk powder, no fat could be demonstrated in the kidneys. These findings are similar to the results obtained with rats. The kidneys of pigs from the lots receiving no skim milk powder, as well as those receiving 2 per cent and 5 per cent skim milk powder, were of a much lighter color than those from the pigs receiving 50 per cent. Histologically, the first three showed a hydropic cytoplasm of the renal epithelium, while that on ration 4 was more homogeneous. The livers presented cytologic changes analogous to those found in the kidneys.

The vertebrae and femurs of pigs receiving rations 1 and 2 were very soft and cut readily with rib shears. Those on ration 3 offered slightly more resistance, while those of the fourth group were extremely

TABLE I—Gains on four experimental rations.

RATION*	Ca	P	Ca/P	DIG. PROTEIN	FAT	AVER. GAINS 122 DAYS (LBS.)
1	.6,098	.2,744	2.2/1	6.96	3.83	18.5
2	.6,344	.2,880	2.2/1	7.48	3.75	25.5
3	.6,713	.3,084	2.14/1	8.25	3.66	33.7
4	1.2,248	.6,144	1.99/1	19.96	2.25	150.0

*Ration 1 contained 98 parts yellow corn but no skim milk powder; ration 2, 96 parts yellow corn and two parts skim milk powder; ration 3, 93 parts yellow corn and five parts skim milk powder; ration 4, 48 parts yellow corn and 50 parts skim milk powder. All rations contained 1.5 parts calcium carbonate and 0.5 part sodium chloride. Each 100 pounds included 15 gm. of ferric chloride and 1.3 cc. of a 20 per cent solution of potassium iodide.

hard and could be cut only with a hand axe. Microscopic examination revealed a rarefied bone formation in the pigs fed rations 1 and 2. In group 3 the trabeculae were somewhat heavier. Those of group 4 presented a very heavy bone formation.

Pig 766, on ration 1, was a gilt. The pigs receiving rations 2, 3 and 4 were males. Pig 767, on ration 2, showed marked tubular degeneration of the testicles and what appeared to be excessive interstitial cells for its age (fig. 1). Pig 768, on ration 3, showed slightly less tubular degeneration and less interstitial cells. Pig 769, on ration 4, showed much less tubular degeneration and only about one-fifth the number of interstitial cells. None of the glands could be considered normal.

TABLE II—Postmortem findings in pigs fed experimental rations.

Pig	Ration	Days on Ration	Marchi Method for Fat		
			Spinal Cord	Liver	Kidney
766	1 (0 S.M.P.)	123	++	+*	+
767	2 (2% S.M.P.)	118	++	+±	±
768	3 (5% S.M.P.)	118	++	+±	T*
769	4 (50% S.M.P.)	118	++	0	0

*Trace.

TABLE III—Spinal cord demyelination and fat in kidneys and liver of cebione-injected pigs.

Pig	Ration	S. M. P. (%)	Cebione	Days on Ration	Spinal Cord	Liver	Kidney
776	1	0	0	159	+++	++	++
775	1	0	+	159	++	++	++
778	2	2	0	154	++	++	++
777	2	2	+	154	++	++	++
780	3	5	0	154	++	+	+
779	3	5	+	154	++	+	+
782	4	50	0	154	++	0	0
781	4	50	+	154	++	0	0

TABLE IV—Postmortem findings on six pigs fed experimental rations over a period of nine months.

Pig	Ration	Days on Ration	Weight (Lbs.)	Marchi Method for Fat		
				Spinal Cord	Kidney	Liver
852	3	278	110	+++	T	T
853	(5% S.M.P.)		86	+*	T	T
849	4		315	+++	0	0
854	(50% S.M.P.)		294	++†	T	T
850	2 (2% S.M.P.)	141	148	+++	T	0
851	4 (50% S.M.P.)	137	147	+++	T	0

*Trace.
†Resorption?

The pigs on rations 1, 2 and 3 showed low stretched acinal epithelium of the thyroid. That from the pig on ration 3 did not appear quite as exaggerated as the first two. The thyroid epithelium of the pig on ration 4 presented a contrast to the first three lots. In this case the epithelium

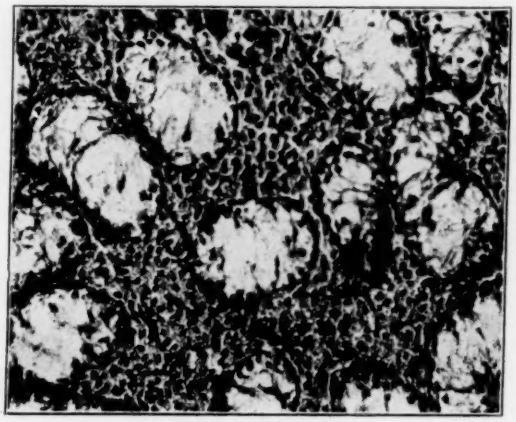


Fig. 1. Testicle, pig 767, which was fed skim milk on ration 2, showing tubular degeneration. Hematoxylin and eosin (x 140).

was higher and the acini much smaller. This might be related to the increased metabolic rate, the body weight gains being about five times as great in lot 4 when the tissues were taken.

About one month subsequent to the destruction of the four pigs described (table II), eight additional pigs were sacrificed for study. These consisted of two animals from each lot or ration, one pig of which had received weekly injections of cebione while the other had not. The results are indicated in part in table III. No difference was found in the intensity of the spinal cord demyelination or in the amount of fat present in the liver or kidneys of cebione-injected pigs and those not injected. (See figures 2 and 3.)

As in the case of the pigs destroyed one month previously, the liver and kidneys of pigs 782 and 781 on ration 4 (table III) were negative for fat. With the lapse of time, the degree of fatty degeneration had increased in the livers of lots 1, 2 and 3 (table III). The adrenals showed no significant variations. Unlike the first group (table II) the thyroid glands of these pigs (table III) failed to show histologic differences, *i.e.*, the thyroids of pigs on ration 4 were not different from those on rations 1, 2 and 3. Degenerative changes in the ovaries and testicles were less advanced in ration 4 than in rations 1, 2 and 3.

Six other pigs were held on some of these experimental rations for about nine months (table IV). Pigs 852 and 853 were continued on ration 3, containing 5 per cent skim milk powder, throughout this period (278 days). Pigs 849 and 854 were fed ration 4, containing 50 per cent skim milk powder, for 278 days. Pigs 850 and 851 of lot 2 received 2 per cent skim milk powder for 141 days and then were changed to ration 4, which was fed for 137 days. With the exceptions of pigs 854 and 853, the spinal cords showed more severe degeneration of the myelin sheaths than the groups killed previously. The space previously filled with fat or myelin in some fibers appeared empty. The findings in these cases and in some previous experiments suggested the possibility of resorption. Pig 849 showed complete absence of fat in the liver and kidneys, while its mate (854) showed a trace.

Pigs 852 and 853 (on 5 per cent skim milk powder) showed a trace of fat in both liver and kidneys. In these, apparently, most of the fat had been removed after prolonged feeding of this ration because pigs on this ration, killed after four months' feeding, showed a significant amount of fat in both liver and kidneys (tables II and III).

The thyroids of the pigs held on these experimental rations for 278 days, as indicated in table IV, showed practically no differences in contrast to the pigs killed earlier. In general, the acinal epithelium was low and flat, with a few acini having low cuboidal cells. In this group (table IV) definite degenerative changes appeared in the ovaries of all. The boar (849) showed advanced tubular degeneration and excessive interstitial cells for an animal of its age. Although this boar was in the same lot with the gilts (854, 850 and 851), they failed to conceive. Pigs 849 and 854, which had received ration 4 containing 50 per cent

skim milk powder, showed a heavier bone formation than the others. The thymus glands throughout the series were uniformly large. The cortex was well developed and none showed evidence of fat or beginning atrophy.

DISCUSSION

The findings in this series of pigs, fed the four experimental rations under dry lot conditions, indicate that the protein factor and vitamin C are not directly responsible for the spinal cord demyelination. On the basis of this experiment and previous investigations, the absence of vitamins A, B, D and E apparently are not the specific causal factors of this degenerative process. Some factor aside from the known vitamins is necessary to prevent demyelination. This factor is present in the green feeds.

The qualitative and quantitative character of the protein in the ration bears a relationship to the fatty degenerations found in the kidneys. The same factor appears to be responsible for the fatty degeneration present in the liver.

Several of the pigs on ration 4 (containing 50 per cent skim milk powder) gained 1.2 pounds per day, with an average of 1.1 for the group. Notwithstanding the rapid growth, the animals showed a slightly stilted gait. They lacked the finish or smoothness that should characterize a normal pig making these gains. Furthermore, the sex organs of male and females showed severe degenerative changes, suggesting the inability to breed which was confirmed by the absence of conception. The myelin sheath degenerations occurred notwithstanding apparently adequate intake of vitamins A, B, C, D, E and G. Some factor in addition to those mentioned is necessary for normal development. This factor, or factors, is present in the grasses or green stuffs. From a practical standpoint the results emphasize the well known but frequently neglected requirement of pasture or high grade alfalfa for the breeding stock and growing pigs.

Until additional nutritional factors are studied and understood in this connection, we are inclined to interpret the myelin sheath degeneration as a general manifestation of malnutrition rather than a change due to a specific cause. The lack of green stuffs during the early life of the pig has

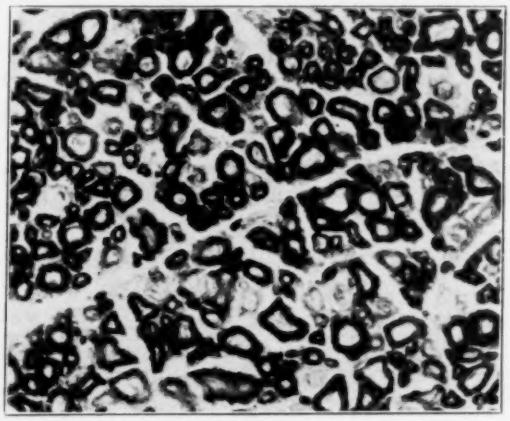


Fig. 2. Spinal cord of pig 776, showing myelin degeneration after being on ration 1 for five months. Marchi method (x 280).

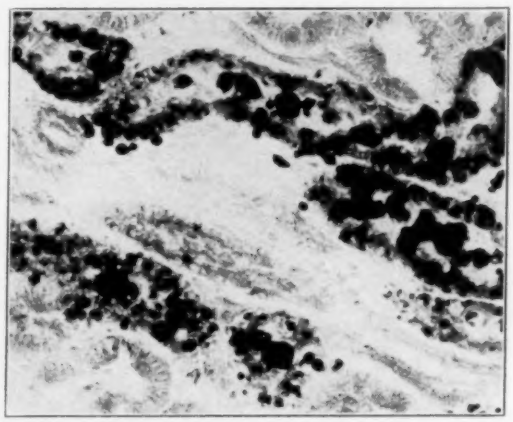


Fig. 3. Kidney, pig 776. Note fatty degeneration. Marchi method (x 280).

a more serious effect than after the rapid growing period. Many unthrifty animals, affected with low-grade enteric diseases and parasites and kept on constantly used dry lots, suffer in part from nutritional disorders, notwithstanding the fact that they receive rations that are beyond criticism from the standpoint of minerals, carbohydrates, proteins and the known vitamins. When such animals are moved to clean pastures in an attempt to overcome the poor sanitation, rapid improvement is often noted. To a great extent this improvement should be attributed to better nutrition.

Treatment of Fox Distemper

By GEORGE L. OTT, Ph. D.
Thiensville, Wis.

THE DOMESTICATION of silver foxes for the production of pelts, being a relatively new industry, involves many unsolved problems, one of the most important of which is disease control. In regard to disease problems, not only is the fur rancher and coöperating veterinarian frequently handicapped by lack of information in respect to the symptoms produced in the silver fox by various etiological agents, but also by lack of information as to the effectiveness of various prophylactic measures.

Fig. 1. Portion of breeding pen for silver foxes on the Nieman ranch.

Usually when an epizoötic appears, a prompt diagnosis must be made and treatment prescribed. Primarily because of economic reasons, there is often little opportunity to evaluate the treatment prescribed by comparison with untreated control animals. Information concerning the value of various biological agents obtained under these conditions does not permit the drawing of accurate conclusions, with the result that the inconclusive information is of little value for future reference. Not only must the treatment be controlled properly by comparison with untreated animals but also a sufficient number of animals under identical conditions must be available so as to reduce chance variation to a minimum.

Herbert A. Nieman & Co., of Thiensville, Wis., and their affiliated ranches have afforded an excellent opportunity of studying, under identical conditions, the effect of certain controlled prophylactic measures against silver fox distemper. Several thousand animals were used.

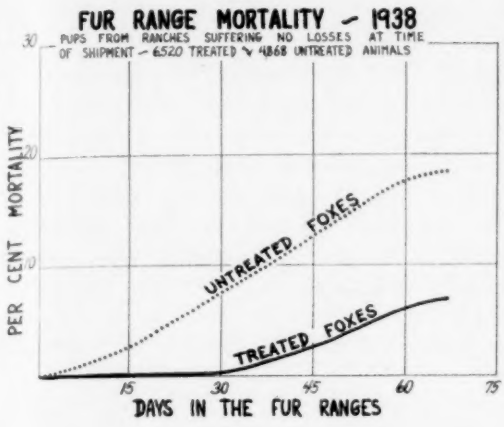
Under regular breeding-pen conditions (fig. 1), the foxes are confined by families, consisting of one pair of breeders and their offspring, to pens of 50′ x 50′. As each family is fed and watered separately, the opportunity of contacting sick animals is greatly reduced. Distemper epizoötics do not occur regularly in the breeding pens, but when they do occur, they run a protracted course.

Each fall, during October, those foxes which are to be pelted are shipped from the home-breeding pens to the Hermansville, Mich., fur ranges. These ranges adjoin one another and are uniform in size, each range consisting of 20 acres of heavily wooded land. Approximately 700 to 750 animals are placed in each range. Under these conditions of intimate contact, *i. e.*, common feeding grounds, fighting, etc., it is readily apparent that opportunities for the spread of disease are markedly increased.

The pups in these fur ranges suffered a considerable mortality for several years from some disease or diseases. These epizoötics, which incurred an average mortality of 35 to 60 per cent, occurred regularly every year during the months of October, November, and December, soon after the pups had been confined to the fur ranges. An intensive survey of the 1937 fur-range epizoötic revealed that distemper was the major disease responsible for these losses.

Bearing in mind the incubation period of the disease and the cost of treatment, a series of controlled experiments with the use of a commercial homologous anti-canine

CHART 1



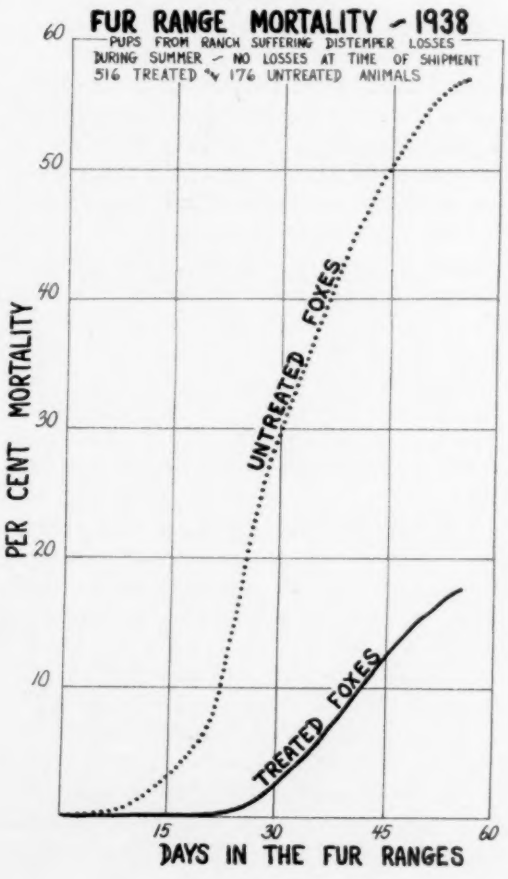
distemper serum* were planned for the 1938 fur-range season. Only foxes born the preceding spring were placed on experiment. Each animal was completely identified by numbers tattooed in the ears. In order to eliminate the possibility of having either a very susceptible or a very resistant litter fall entirely into either the treated or untreated groups, treatments were given at random within litters. All treated animals were given a dose of 10 cc. of homologous anti-canine distemper serum injected subcutaneously into either axillary space at the time of shipment to the fur ranges during the month of October. Treated foxes were not segregated but were mixed with untreated foxes in each range. Each range was filled within two days. All of the foxes were fed the standard diet of horse meat, cereal, minerals, etc. A total of 19,349 animals were placed on experiment.

The foxes in the 1938 experiments may be divided into groups coming from three sources: (1) from ranches suffering no losses prior to or at the time of shipment; (2) from a ranch which had suffered an epizoötic of distemper during the summer of 1938 and which was controlled effectively with homologous anti-canine distemper serum with no subsequent losses prior to or at the time of shipment, and (3) from a ranch where losses from distemper and its complications were occurring at the time of shipment.

Chart I shows the mortality per cent among 6,520 treated and 4,868 untreated animals from ranches suffering no losses prior to or at the time of shipment (group I). The epizoötic among these animals began about ten days after the foxes had been confined to the fur ranges. The treated foxes showed a passive immunity lasting for a period of approximately 30 days. When losses began in the treated group, they occurred at a lower rate than the losses in the untreated control group. Over a period of 67 days, or at the time of final pelting, the treated group suffered a final mortality of 6.7 per cent, as compared to a 17.8 per cent in the untreated group over the same period of time.

Chart II shows the mortality per cent among 516 treated and 176 untreated animals from a ranch where distemper had been prevalent during the summer of 1938 and which had been controlled effectively with homologous anti-canine distemper

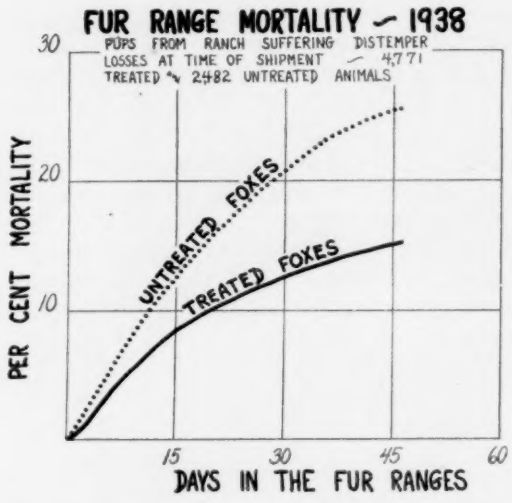
CHART 2



*Serum used was prepared by actively hyperimmunizing dogs against live canine-distemper virus.

serum with no further losses prior to or at the time of shipment (group II). The epizoötic among these animals began about 15 days after the foxes had been confined to the fur range. The treated foxes showed a passive immunity for a period of 28 days

CHART 3



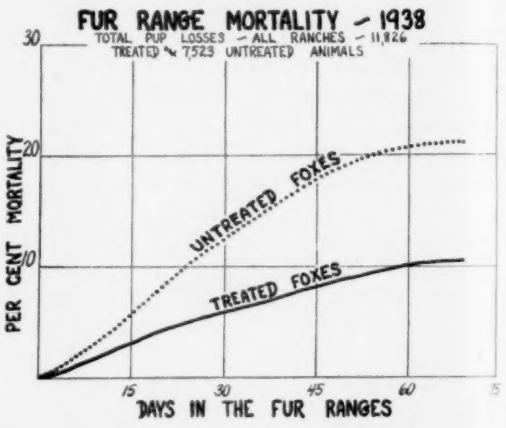
before losses began as well as a decreased rate in mortality as the epizoötic continued. At the time of final pelting the treated group suffered a final mortality of 17.2 per cent as compared to the final rate of 57.2 per cent in the untreated group.

Chart III shows the mortality per cent among 4,771 treated and 2,482 untreated animals from a ranch which, as previously indicated, was suffering severe losses from distemper and its complications at the time of shipment (group III). The value of the treatment is evidenced by the immediate, lower death rate of the treated foxes, which was sustained until the animals were pelted, or a period of 47 days, with a final mortality of 15.3 per cent. The untreated control animals in this series suffered a much higher death rate with a final loss of 25.7 per cent.

Chart IV is a summary of the total mortality per cent among 11,826 treated and 7,523 untreated foxes on experiment, regardless of ranch origin, *i. e.*, of all foxes in the 1938 experiments. The total treated group showed not only a marked decrease in the mortality per cent but also a reduction in the rate of mortality following serum treatment for a period as long as two months, or until the animals were pelted. The entire treated group up to the time of pelting showed a final mortality of 10.5 per cent against a final one of 21.1 per cent of the untreated controls.

The results obtained during the 1938 fur-range season indicate that the commercial homologous anti-canine distemper serum used was effective in reducing the losses incurred by distemper in the silver fox. All of the treated and untreated experimental animals of the same age were under identical conditions of environment, weather and feed. A random selection of animals

CHART 4



for treatment within litters eliminated the possibility of having either resistant or susceptible litters fall entirely into either the treated or untreated groups. In all cases, the mortality of the treated groups was markedly lower than that of the untreated, or control, animals in the same fur ranges, the final mortalities of the treated animals ranging from one-third to one-half of that of the controls.

Among animals with no losses occurring prior to or at the time of shipment, the epizoötic began within ten to 15 days after the time of shipment. In these same ranges, the period of passive immunity conferred by the use of anti-canine distemper serum was from 28 to 30 days. Similarly, the beneficial effects of the serum were ob-

(Continued on next page.)

A Single-Dip Stain for the Direct Examination of Milk*

By JEAN BROADHURST and CHARLES PALEY
Teachers College, Columbia University, New York, N. Y.

THIS STAIN was developed for making direct microscopic counts of milk, employing the technic ordinarily used in direct microscopic examination. Like the Newman stain, it combines fat extraction, fixing and staining.

The advantage of this stain over other methylene-blue stains is that only the organisms and other cells take the deep blue stain, while the background stains a very faint pink. This permits the counting of all organisms and shows their characteristic formation. The nuclei of the white blood cells and tissue cells take a deep blue stain, while the cytoplasm takes a faint blue. The advantages are as follows:

1. The differential background permits the counting of all organisms and shows their formations.

2. This stain permits easy differentiation between bacteria and all other cells and foreign bodies, such as precipitated dye, amorphous bodies, etc.

3. Eyestrain in routine work is eliminated because the organisms do not have to be searched for and are not hidden by darkly stained, congealed serum solids on the smear.

4. The serum solids on a properly made smear are stained a faint pink.

5. Preparation of the stain is simple, since the reagents are easily obtainable.

6. No decolorizing of a heavily stained smear is necessary, a procedure which sometimes decolorizes organisms as well as the background, thereby causing the possibility of low counts.

PREPARATION OF STAIN

To 54 cc. of 95 per cent ethyl alcohol is added 0.4 cc. of concentrated sulphuric acid (c.p.). This solution is mixed with 40 cc. of technical tetrachlorethane in a flask and heated to about 55° C. (no higher). Add the combined solution while hot to from 1 gm. to 1.2 gm. of methylene blue dye, shaking vigorously until the dye is as completely dissolved as possible. Next, add 8 cc. of a 1 per cent alcoholic solution of fuchsin (prepared by dissolving 1 gm. basic red fuchsin in 100 cc. of 95 per cent ethyl alcohol). The stain should be mixed well, cooled, filtered and kept in tightly stoppered bottles.

In the preparation of the stain a pipette calibrated in 0.1 cc. should be used for measuring the sulphuric acid, since too much acid may result in too faint a background, while too little may cause difficulty in washing the blue out of the smear.

DIRECTIONS FOR USE

1. Prepare the milk smear by using 0.01 cc. of milk spread over 1 sq. cm. of surface, as is the general procedure for making bacteria counts by the direct microscopic method. (2 sq. cm. may be covered, if it is desired.)

2. Dry the smear on a flat surface in a warm place within five minutes.

3. When dry, dip into the stain for about 15 seconds, or flood the smear for about 15 seconds; drain off excess stain and dry while flat in a warm place.

*At the New York meeting of the Association, a paper entitled, "A Differential Stain for the Direct Examination of Milk," was presented by the authors. Since that time, the single-dip stain has been adopted officially by the New York City Board of Health.

(*Continued from page* 524)

served in those animals among which the epizoötic already was in progress at the time of shipment. It must be remembered that the use of serum confers a passive immunity, affording only a temporary protection. Under the above controlled conditions with several thousand animals, however, the 1938 experiments indicate that the use of homologous anti-canine distemper serum as a prophylactic agent against fox distemper is an economically sound measure.

4. Wash in cold water until all the blue is washed out of the smear and the smear assumes a faint pink color.

5. Dry, and examine under oil immersion.

Because the organisms stand out so readily in contrast to the background, much higher counts are obtained by the use of this stain than with other methylene-blue stains. It should be noted that the method is actually the same as that employed by those using the Standard Methods of Milk Analysis, except that a different stain is used. Additionally, it may be well to remark that where a smear is unevenly made, *i.e.*, where some parts are thicker than others, it may be difficult to wash out all the blue from the thickened parts of the smear. However, this does not interfere in any way with the counting of the organisms and the easy differentiation of them. In the smear of 2 sq. cm., all the blue is washed out perfectly.

Famous Homing Pigeon

"Old Anchor" was a fine type of homing pigeon used by the signal corps of the German army during the World War. He

—*A.P. Wire Photo.*

Old Anchor

alighted on an American destroyer in British waters in 1918, and thereafter was a ward of the U. S. navy—a prisoner of war. This famous Columbidae died the other day at the ripe old age of 21 and was buried at Monmouth, New Jersey, with military honors.

Medical Problems of the Hour

A glance through the program for the Saint Louis session of the American Medical Association, May 15-19, 1939, gives an idea of the topics engaging the attention of medical science. The subjects of current interest in our branch of medicine which catch the eye are (1) the uses of sulfanilamide and its derivatives; (2) the newer work on vitamins, particularly nicotinic acid and the vitamin B complex; (3) the developments in endocrinology and the use of the new endocrine products; and (4) the progress in the treatment of pneumonia, especially pneumonia of infancy.

French World War Physicians

The Paris correspondent to the *Journal of the American Medical Association* (Aug. 13, 1938), in reporting the unveiling of a monument to the memory of World War physicians, writes that out of the 22,000 physicians practicing in France when the war broke out in 1914, 18,000 were mobilized. At the time of the armistice there were 21,181 physicians in the sanitary corps, which comprised, in addition, 3,821 pharmacists, 1,000 dentists, 5,238 hospital officials, 120,000 male nurses, and 600 female nurses.

The mortality was as follows: physicians, 2,108; pharmacists, 321; dentists, 142; hospital officials, 500; male nurses, 9,213; and female nurses, 132.

EDITORIAL

Electrically Induced Ejaculations*

THE USE of an electric current passed along the genital nerves to induce spontaneous ejaculation was first practiced by Gunn[1] in Australia and it has been popular in England, Italy and Spain for some years.

The best article on the subject we can locate at this moment was published in Belgium, in 1938. It furnishes us with the information contained herein.

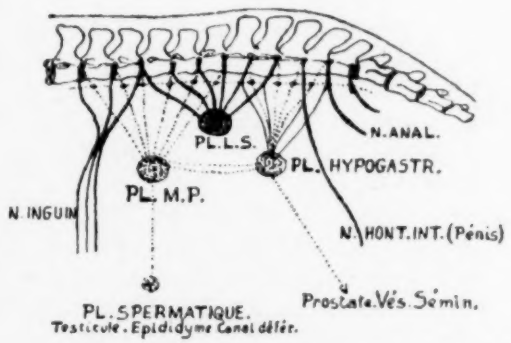
The advantages of the method are said to lie in the facts that the ejaculation is more natural than one induced by other methods, that the semen collected is less apt to be contaminated, and that by properly handling the electrode at the loin, one can get either concentrated semen, normal semen, or semen diluted with seminal and prostatic fluid.

The principle involved is that of exciting rhythmic contractions by sending a current along the genital nerves from electrode to electrode, one in the rectum and the other tapping upon the lumbar region. After a certain number of shocks (excitations) ejaculation occurs and the semen can be collected at the penial meatus. The semen thus taken is said to be favorable for the preservation of the spermatozoa it contains.

As the muscles of the genital apparatus are governed by the nervous system of the lumbo-sacral portion of the cord, an anatomo-physiological study of the region reveals the mechanism of the method.

The lumbar portion of the cord gives off six pairs of spinal nerves. The inferior branches of the first three of these furnish the nervous filaments forming the external and internal inguinal nerves—the nerve supply of the cremaster, the tunica vaginalis, the scrotum and the sheath. The inferior branches of the fourth, fifth and sixth pairs of the lumbar nerves join with the first two pairs of sacral nerves to form the lumbo-sacral plexus, the posterior gluteal branch of which gives a filament to reinforce the pudic nerve. The pudic nerve, derived from the third pair of sacral nerves, supplies the penis, the ischial muscles and the bulbo-cavernosum.

Moreover, the portion of the great sympathetic nerve, whose afferent branches in this region are derived from the inferior branches of the lumbar nerves, forms the posterior mesenteric plexus from its efferent branches. The posterior mesenteric



—*From Annales de Médecine Vétérinaire.*

THE INNERVATION OF THE GENITAL APPARATUS OF THE MALE

PL.L.S., lumbo-sacral plexus; N.ANAL., anal nerve; PL. HYPOGASTR., pelvic plexus; PL.M.P., posterior mesenteric plexus; N.INGUIN., inguinal nerve; N.HONT.INT. (Pénis), internal pudic nerve; PL. SPERMATIQUE, spermatic plexus; Testicule, testicle; Epididyme, epididymus; Canal défér., vas deferens; Prostate, prostate; Vés. Sémin., seminal vesicle.

plexus through its spermatic portion supplies innervation to the testicles, the epididymus and vas deferens. The sacral portion of the great sympathetic nerve forms the pelvic or hypogastric plexus, which governs the seminal vesicles and prostate.

By electrically exciting the anterior lumbar nerves, Gunn (*loc. cit.*) obtained pure

*Excerpt from L'insemination artificielle chez les animaux domestiques, by A. Duvuyst, Annales de Médecine Vétérinaire, August-September, 1938.

[1]Cited by the author, *loc. cit.*

semen but observed that when the lumbosacral plexus was excited the issue contained seminal and prostatic fluid, showing that the collection of semen by this method is based upon the facts described and illustrated above.

The technics of the procedure are described as follows: The two electrodes are impregnated with an electrolyte composed of sodium chloride, 10 per cent. They are covered with chamois. The regular needle is replaced by a roulette electrode. When one of the electrodes is set in the rectum the other is rolled with a tapping action over the loin ten to 15 times at intervals of several moments. An alternating current with a voltage potential of 30 is used.

The author points out that indirect methods of collecting semen are objectionable because the semen is not pure and the ejaculation incomplete while the direct method is a practical imitation approaching natural conditions.

Since this procedure has been highly honored for its value in rams and bulls, it would seem to be a popular method among dog breeders who, we are informed, are becoming increasingly interested in artificial insemination.

Artificial insemination was practiced by Arab tribes for many centuries, but it was never generally employed systematically in any country until recent years. Forty years of study by Elie Ivanow at the Institute of Animal Industry of Russia brought renewed interest in the practice.

A New Activity

AMONG the new activities of the Association is that of aiding the junior chapters at the various colleges in obtaining outside speakers for their meetings. In April, the executive secretary addressed the students of Kansas State College on veterinary deontology and, in May, H. E. Moskey, of the Committee on Proprietary Pharmaceuticals, will tell the chapter at the University of Pennsylvania about the provisions of the new food and drugs law, which is destined to play an important part in the therapeutic drama of the future.

The practice of bringing in selected speakers to address the undergraduates on selected subjects is a wise departure complimentary to the deans.

Manuscripts for Publication

IT WILL be necessary in the future to exercise greater care in selecting manuscripts for publication in the JOURNAL. With the aid and advice of the Reorganizing Committee and the associate editors, certain requirements will be adopted in regard to such important matters as (1) length of titles and text; (2) size, number and value of illustrations; (3) number and importance of tables and charts; (4) revealing legends for illustrations, especially for photomicrographs; (5) unnecessary words, phrases, clauses, sentences and even paragraphs, requiring editorial pruning; and (6) last but not least, modern usage in punctuating, abbreviating, capitalizing, italicizing, etc., requiring avoidable editorial work.

Nota bene, that these include only matters of current expediency. Set rules on the adjudication of the value and merit of scientific papers are delicate questions for future action. Whether scientific articles embody principles of tremendous value or reek with fanciful fiction may be a matter of reversible opinion. The wildest theory of today may be the glorified fact of the morrow. Hence, in this respect the JOURNAL will continue to judge manuscripts by their source and will strive to make room for them, provided the authors will keep our mechanical and editorial capacity in mind.

One thing is now certain, a journal of 128 pages should be the minimum, not the optimum objective of the Association, for when the proposed new departments are installed, deflation back to 96 pages will be unthinkable. As formerly, the authors of original scientific articles would have to await attention indefinitely and the plan to add popular departments would have to be abandoned.

The Endocrine Complex

THIS BRIEF is intended to answer a number of inquiries requesting information as to the therapeutic value of certain endocrine products, widely advertised and extensively employed in clinical work. In view of the "newness of things" in that field, plus the importance of them, particularly in animal production, it is not reasonable either to extol too highly or criticize prematurely a departure as promising as the use of hormones.

Nothing more bewildering has ever come into the practice of medicine than the hormones. The reason is understandable, since the science called endocrinology is a relatively new branch of physiology that dips deeply into the ways of Nature. If it frustrates the scientist and confuses the practitioner, the cause is the sea of unknown that may always keep medicine pretty close to the line of scrimmage. As a noted authority has said, endocrinology is a fascinating tale without a climax, and this we repeat in order to prevent one's discouragement which is brought about by the deluge of opinions and ideas on hormones that must somehow be reduced to common sense in the practice of medicine.

Contradictory researches on any one of the known glands of internal secretion (there are probably many yet to be found) are sufficient to cite as examples of this undeveloped study. In short, endocrinology is an infant, but one that is perhaps capable of becoming a healthy giant. In so far as its therapeutic application is concerned, the worst that anyone could say about it is that it is something to seize as an excellent prospect, and judge in the future with an open mind.

Taking the pituitary as the theme, as the story now goes, we have the headquarters of the entire endocrine system, where all direction is sent out to the lesser units of the chain. Thyroxin, insulin, adrenalin, folliculin, to name but the well-knowns of the group, are governed from these headquarters. As these include about everything worth while in physiology (metabolism, energy, reproduction), the study of the pituitary, or hypophysis, as some prefer, is truly profound. This tiny organ is said to govern almost everything from its cranial P.C., but beyond that post the plot begins to thicken. Soon, the units of the chain begin to work one against the other. What one stimulates the other inhibits; what one energizes the other depresses. The hypoglycemic Islands of Langerhans work against the hyperglycemic adrenals and, between the two, the normal level of blood sugars is maintained. Comparable pressors and depressors are found in the gonads and other organs of reproduction.

Blood-sugar regulation is, however, the best example of the complex, since it is the one function of the endocrine system that is fairly well understood. Insulin lowers blood-sugar levels through several of its properties. It mobilizes the carbohydrate reserve and fans the combustion of sugars while simultaneously depressing their production (Kepinov, 1938). On the other hand, adrenalin raises the blood-sugar levels by drawing upon the hepatic and muscular glycogen. In other words, through this antagonism, gycemia is regulated. But the mechanism is not nearly that simple.

When hypothysectomized, dogs develop hyperadrenalemia and hypoglycemia, with a tendency to convulsions. Adrenalin alone, therefore, does not cause hyperglycemia. To accomplish that result (hyperglycemia) the pituitary must contribute its aid. In fact, three internal secretions coöperate in maintaining blood-sugar levels, namely: insulin, which tends to lower the level, and the pituitary-adrenal combination, which raises it.

Inasmuch as these "coöperations and antagonisms" prevail throughout the system of internal secretion, this example is cited only to show the complexity of endocrinology in the practice of medicine. No part of the study can be clarified in abbreviated form.

Executive Board Elections

ALTHOUGH the nominations do not close until the 28th of April, which is a few days after this issue goes to press, the probable candidates will be as follows. This announcement is not premature because the voting will have closed before this issue reaches the readers. The ballots for the election of one of these candidates will be distributed in a few days.

District 4

COTTON, W. E., Auburn, Ala.

Member of faculty of the Alabama Polytechnic Institute. Graduate of the George Washington University, 1911. Joined 1911. Former Pathologist of the B.A.I. Research Laboratory.

DIMOCK, W. W., Lexington, Ky.

Professor of Veterinary Science, College of Agriculture, University of Kentucky. Graduate of New York State Veterinary College, Cornell University, 1905. Joined 1906. Resident secretary for Iowa, 1910-1913; resident secretary for Kentucky, 1922-1924; member of Executive Board, District 4, 1935.

GILLMANN, JOHN H., Memphis, Tenn.

Distributor, Jensen-Salsbery Laboratories. Graduate of Saint Joseph Veterinary College, 1917. Joined 1926. Third vice-president, 1938-1939; chairman of the Committee on Local Arrangements for Memphis meeting, 1939.

MCADORY, I. S., Auburn, Ala.

Dean, Division of Veterinary Medicine, Alabama Polytechnic Institute. Graduate of McKillip Veterinary College, 1908. Joined 1908. State Veterinarian of Alabama.

MOORE, WILLIAM, Raleigh, N. C.

State veterinarian of North Carolina. Graduate of U. S. College of Veterinary Surgeons, 1911. Joined 1926. Resident secretary for North Carolina, 1926-1933; member of Executive Board, District 4, 1935-.

District 10

BRUMLEY, O. V., Columbus, Ohio

Dean, College of Veterinary Medicine, Ohio State University. Graduate of Ohio State University, 1897. Joined 1919. Past president, 1937-1938; member of Executive Board, District 10, 1930-1936; member of Reorganization Committee, 1939.

HALLMAN, E. T., East Lansing, Mich.

Professor of Animal Pathology, Michigan State College. Graduate of Alabama Polytechnic Institute, 1910. Joined 1915. Resident secretary for Michigan, 1919-1921.

HAYS, C. H., Lansing, Mich.

Inspector-in-charge, Michigan State Bureau of Animal Industry. Graduate of Ohio State University, 1908. Joined 1916. Member of Executive Board, District 7, 1930-37 (resigned because of change of address); state veterinarian for Michigan.

KILLHAM, B. J., East Lansing, Mich.

Extension specialist in animal diseases, Michigan State College. Graduate of McKillip Veterinary College, 1912. Joined 1917. Resident secretary for Michigan, 1921-1922, 1924-1927, 1931-1933; former state veterinarian for Michigan.

ZIMMER, F. A., Pataskala, Ohio

Practitioner. Graduate of Ohio State University, 1909. Joined 1919. Former state veterinarian of Ohio; member of the Executive Board, District 10, 1937-.

Are You Interested Too?

THE REMARKABLE interest that is being taken in the development of veterinary medicine in the various countries now dominating the affairs of this period should be a hint to veterinarians who fail to participate in organized veterinary medicine.

In their determination to become or remain self-sufficient, independent sovereignties, the utilitarian work of trained veterinarians is attracting outstanding attention throughout the great nations of the world and, as a consequence, the societies devoted to the study and use of animal medicine are feeling the effect of the augmented interest in what they are doing. The national associations of Great Britain, France, Germany, Japan, Russia and others, as well as that of the United States and Canada, have the public eye focused upon them, contrary to the unnoticed organizations they once were—weak, struggling, undaunted, hoping for a little attention to what they were attempting to accomplish. Thanks to those who have labored, through knowledge of disease, to provide their countries with a systematically policed animal industry, veterinary medicine is now being rated at par value for the first time in modern history.

If you are interested, you are probably a member. If not, your colleagues are sorry for the difficulties they still have to overcome.

APPLICATIONS

NEVER before has the prospect of an increased membership been quite so promising. *And no wonder!* For, the following list of 119 applicants represent the largest number ever to be enrolled in any one month in the entire history of the Association.

First Listing

(See January, 1939, JOURNAL)

ABBOTT, JOHN E.
1519 Poyntz Ave., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

ADAMS, CLARENCE LINWOOD
154 Main St., Danielson, Conn.
D. V. S., American Veterinary College, 1896. Vouchers: Geo. E. Corwin and R. L. Smith.

ALSON, MARION C.
1214 Bluemont, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

ANDERSON, D. WARNER
Route 1, Box 7, Eaton, Colo.
D. V. M., Colorado State College, 1939. Vouchers: Frank Thorp, Jr., and A. W. Deem.

ARCHIBALD, RYLAND MCGREGOR, JR.
890 Prince St., Truro, Nova Scotia, Can.
B. V. Sc., Ontario Veterinary College, 1937. Vouchers: W. J. R. Fowler and R. A. McIntosh.

BEBKOWITZ, ISRAEL
487 Ridgewood Ave., Brooklyn, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

BLOOD, BEN
920 Woodford Ave., Fort Collins, Colo.
D. V. M., Colorado State College, 1939. Vouchers: Frank Thorp, Jr., and G. S. Harshfield.

BOLKS, HERBERT PAUL
Hull, Iowa.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

BRINKER, WADE OBERLIN
901 Kearney St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

BURDO, ANTHONY M.
723 Laramie, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

BURR, FRANKLIN HAROLD
247 Audley St., So. Orange, N. J.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

CASSELBERRY, NORWOOD H.
604 N. 5th St., Savanna, Ill.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

CLARK, FORREST W.
Veterinary Hospital, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

CODDINGTON, ALTON MONROE
Alexander, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

COHEN, GEORGE
25 Cass St., Springfield, Mass.
D. V. M., Alabama Polytechnic Institute, 1937. Vouchers: Lt. Col. J. F. Crosby and Max H. Carlin.

COLLINS, WAYNE DEVERE
600 N. 15th St., Marysville, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

CONRAD, HARRY JACOB
1214 Bluemont, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

CORCORAN, JOHN ROBERT
801 Remington, Fort Collins, Colo.
D. V. M., Colorado State College, 1939. Vouchers: A. W. Deem and G. S. Harshfield.

CREECH, GILBERT T.
1408 Manchester Lane, N. W., Washington, D. C.
D. V. S., Kansas City Veterinary College, 1910. Vouchers: H. W. Schoening and Wm. M. Mohler.

CURRY, PHILIP H.
2510 N. 11th St., Kansas City, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DAMER, EUGENE FRANCIS
1130 Vattier St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DAVIDSON, W. MAX
307 Holmes St., Huntsville, Ala.
D. V. M., Alabama Polytechnic Institute, 1938. Vouchers: R. L. Mundhenk and W. E. Cotton.

DAVIES, ALBERT A.
3325 Agnes Ave., Kansas City, Mo.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DECKER, CLIFFORD N.
Arlington, Neb.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DECINQUE, PETER
607 N. Manhattan Ave., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DENTON, JOHN PATTON
1130 Vattier, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

DIETERICH, WILLIAM HYDE
1203 Laramie, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

EMERSON, CAPT. HARRY H.
Fort Brady, Sault Ste. Marie, Mich.
D. V. M., Chicago Veterinary College, 1917. Vouchers: Lt. David S. Hasson and Lt. Col. Will C. Griffin.

ESKELDSON, JAMES A.
Ramona, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

EVANS, DAVID EDWARD
1238 S. Third St., Montrose, Colo.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

FELDMAN, MURRAY
3021 E. 7th St., Brooklyn, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

FINKE, CAPT. ELMER FRED
Box 853, Mobile, Ala.
D. V. M., Kansas State College, 1933. Vouchers: Col. B. A. Seeley and L. A. Merillat.

FLADNESS, SEVERIN O.
Bureau of Animal Industry, Washington, D. C.
D. V. M., Chicago Veterinary College, 1912. Vouchers: T. W. Cole and William M. Mohler.

FRANCIS, CHARLES KENNETH
2630 Columbia Pike, Arlington, Va.
B. V. Sc., Ontario Veterinary College, 1937. Vouchers: Charles Gibson Grey and William M. Mohler.

GERMANIO, PETER JOSEPH
Box 94, Belleplain, N. J.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

GREEN, ARTHUR L.
Dandridge, Tenn.
D. V. M., Michigan State College, 1938. Vouchers: D. Coughlin and E. B. Parker.

GROSS, GLENN G.
Russell, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

GROVES, LAWRENCE W.
Office of the Corps Area Surgeon, HFCA., Boston Army Base, Boston, Mass.
B. V. Sc., Ontario Veterinary College, 1938. Vouchers: J. N. McIlnay and Lt. Col. J. F. Crosby.

GUILFOIL, THOMAS J.
221 N. 17th St., Kansas City, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HAMILTON, CLARE C.
340 N. 16th, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HANTMAN, HARRIS WARREN
84 Beaver St., Brooklyn, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HAVLIK, ALBERT LEO
Tampa, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HAYES, ELDON FRANCIS
1315 E. Broadway, Newton, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HAYES, ISAAC EDWARD
1012½ Wellington, Waterloo, Iowa.
D. V. M. Iowa State College, 1937. Vouchers: Edmund W. Burke and Frederick F. Meads.

HENRIKSON, MERLE LOGAN
1725 Anderson, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HERZBERGER, ARTHUR C.
3383 W. 30th Ave., Denver, Colo.
D. V. M., Colorado State College, 1939. Vouchers: A. W. Deem and E. N. Stout.

HINES, CHARLES P.
Wells Hill Road, Lakeville, Conn.
V. M. D., University of Pennsylvania, 1936. Vouchers: Mulford C. Thompson and Geo. E. Corwin.

HOLCOMB, WALTER BROCKETT
Newton, Conn.
V. M. D., University of Pennsylvania, 1937.
Vouchers: Philip H. Gerlach and V. M. Knapp.

HUANG, FUNG KUAN
1215 Thurston St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

HUTTON, AUBREY MEANS
231 Michigan Ave., Saint Joseph, Mo.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

INNES, DONALD C.
411 Saint Vincent St., Philadelphia, Pa.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

IRVIN, CHARLES EARL, JR.
300 E. Elizabeth, Fort Collins, Colo.
D. V. M., Colorado State College, 1939. Vouchers: G. S. Harshfield and Frank Thorp, Jr.

ISBELL, S. B.
Morristown, Tenn.
D. V. M., Alabama Polytechnic Institute, 1935. Vouchers: D. Coughlin and R. L. Mundhenk.

JOKERST, HERMAN A.
Waco, Neb.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

JONES, CARL HERBERT
328 W. Thatch St., Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and W. E. Cotton.

JONES, RAYMOND ALBERT
Penalosa, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

JONES, WILLIAM ALVIN
425 Lamont St., Johnson City, Tenn.
D. V. M., Grand Rapids Veterinary College, 1915. Vouchers: M. Jacob and Robt. L. Hummer.

JORDAN, FRANK WILSON
421 N. 16th St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

KANAWYER, WENDELL LEE
1104 Moro St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

KEY, JOSEPH BOSTON
Route No. 2, Kansas City, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

KNAPPENBERGER, JACK ROSS
221 N. Delaware, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

LABAHN, CHARLES D.
1609 S. Moniteau, Sedalia, Mo.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

LEE, ROBERT JEROME
562 W. 164th St., New York, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

LIEBENGOOD, HOWARD B.
221 N. Delaware Ave., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

LOUGHRIDGE, HENRY
Lyndon, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

LOVE, E. LELAND
724 Kearney St., Manhattan, Kan.
D. V. M., Kansas State College, 1938. Vouchers: Chas. H. Kitselman and E. R. Frank.

MACLEAN, GEORGE JAMES
Route 1, Box 169, Littleton, Colo.
D. V. M., Colorado State College, 1939. Vouchers: A. W. Deem and E. N. Stout.

MALLE, ALBERT LEON
Mulberry, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

MARSTON, EDWARD DEARBORN
224 S. Gay St., Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939.
Vouchers: R. L. Mundhenk and W. E. Cotton.

MASSEY, II, JOSEPH R.
1005 Bluemont, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

MCALLISTER, WILLIAM J.
2115 Anacapa St., Santa Barbara, Calif.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

MCKENNEY, FRANK DONALD
Bureau of Biological Survey, State College of Washington, Pullman, Wash.
D. V. M., Colorado State College, 1928.
Vouchers: E. E. Wegner and J. E. McCoy.

MILLENBRUCK, LT. EDGAR W.
Hdqrs. Missouri District, C.C.C., Fort Leavenworth, Kan.
D. V. M., Kansas State College, 1936. Vouchers: Lt. Col. Geo. J. Rife and R. R. Dykstra.

MILLER, FRANK G.
823-23rd St., Ogden, Utah
D. V. M., Iowa State College, 1908. Vouchers: Irvin Owens and E. D. Leiby.

MILLER, LEONARD JOHN
Clarkson, Nebr.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

MORRISON, GEORGE ALEXANDER
141—1st Ave., Salt Lake City, Utah.
B. S., D. V. M., State College of Washington, 1932. Vouchers: Jean C. Flint and Lt. Col. John W. Miner.

MORTON, L. R.
Toronto, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

MOSSMAN, DONALD FLEET
1130 Vattier St., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

NEBB, SAMUEL S.
775 Blake Ave., Brooklyn, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

NELSON, WALBERT O.
1111 Bluemont Ave., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

NOSSOV, MORRIS AARON
Kansas State College, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

PALEN, JOSEPH S.
105 W. 16th St., Hays, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

PATTERSON, ROY ELLSWORTH
1572 Overton Park, Memphis, Tenn.
D. V. M., Iowa State College, 1923. Vouchers: C. P. Branigan and John H. Gillmann.

PILE, EUGENE FRANK
203 Westwood Court, Lexington, Ky.
D. V. M., Kansas State College, 1916. Vouchers: T. P. Polk and Floyd E. Hull.

PLUMMER, JR., CHAS. BENJAMIN
123 Cox St., Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and J. E. Greene.

POPPENHOUSE, CURTIS ALBERT
416 Laramie, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

POPPENHOUSE, GERHARD C.
416 Laramie, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

PRENDERGAST, BRUCE
Route 1, Box 189, Fort Collins, Colo.
D. V. M., Colorado State College, 1939. Vouchers: A. W. Deem and E. N. Stout.

RAILSBACK, GUY ARTHUR
Langdon, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

RATTRAY, M. J., JR.
114 Thomas St., Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and W. E. Cotton.

REMSBERG, GLEN S.
La Harpe, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

RODABAUGH, DONALD EDWIN
Norborne, Mo.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

ROGERS, ARTHUR BARTLETT
5809 Narcissus Ave., Baltimore, Md.
D. V. M., Cornell University, 1934. Vouchers: H. S. Miller and A. J. Sipos.

ROVNER, BERNARD
1220 W. Columbia Ave., Philadelphia, Pa.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

ROYCROFT, WILLIS CHARLES
Bayou La Batre, Ala.
D. V. M., Alabama Polytechnic Institute, 1916. Vouchers: R. L. Mundhenk and J. E. Greene.

RUBIN, HARVEY L.
Box 481, Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and W. E. Cotton.

RUSH, LOYAL LUTHER
Chanute, Kan.
D. V. M., Kansas State College, 1933. Vouchers: R. A. Coffey and R. R. Dykstra.

SAN MARTIN SAENZ, JULIO J.
Acosta 358, Havana, Cuba.
D. V. M., Havana University, 1909. Vouchers: Bernardo J. Crespo and L. A. Merillat.

SAUNDERS, WILLIAM GLENN
7742 Aberdeen St., Chicago, Ill.
D. V. M., U. S. College of Veterinary Surgeons, 1918. Vouchers: L. A. Merillat and Malcolm J. Thompson.

SCHNEIDER, ARTHUR PHILIP
Box 196, Moscow, Idaho.
B. S., D. V. M., State College of Washington, 1938. Vouchers: E. E. Wegner and E. T. Baker.

SEIBOLD, HERMAN R.
412 Baltimore Blvd., Riverdale, Md.
V. M. D., University of Pennsylvania, 1931. Vouchers: Harry W. Schoening and Wm. M. Mohler.

SHINDELL, MAX MARTIN
105-06 Metropolitan Ave., Forest Hills, N. Y.
D. V. M., Cornell University, 1937. Vouchers: Nathan Wernicoff and B. J. Finkelstein.

STAPLES, WILFRED WARNER
1112 Quintard Ave., Anniston, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and I. S. McAdory.

STEELE, DARRELL STANLEY
615 Leavenworth, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

STOUDENMIRE, WILLIAM FRANK
403 W. Minnesota Ave., Deland, Fla.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

SUTHERLAND, H. L.
State Serum Plant, Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1939. Vouchers: R. L. Mundhenk and James E. Greene.

TANENBAUM, GEORGE GILBERT
930 Montgomery St., Brooklyn, N. Y.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

TAYLOR, EDGAR LEWIS
1214 Bluemont, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

THOMSON, LT. BURTON C.
1921-17th Ave., Columbus, Ga.
D. V. M., Iowa State College, 1938. Vouchers: Lt. Col. Horace S. Eakins and Lt. Col. Chauncey E. Cook.

TODD, ROBERT S.
1214 Bluemont, Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

TURNER, NATHAN ALLEN
Route 2, Grand Junction, Colo.
D. V. M., Colorado State College, 1939. Vouchers: A. W. Deem and E. N. Stout.

VARDIMAN, PHILLIP HARRIS
1005 Bluemont Ave., Manhattan, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

VEST, DON CARROLL
Fort Moultrie, S. Car.
D. V. M., Iowa State College, 1937. Vouchers: Col. B. A. Seeley and J. C. Wright.

VIERHELLER, RALPH C.
211 W. Myrtle, Fort Collins, Colo.
D. V. M., Colorado State College, 1939. Vouchers: G. S. Harshfield and A. W. Deem.

VOLLMAR, ROLAND EMIL FREDRICK
407 Oak Ave. W., Montgomery, Minn.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

WATSON, DOUGLAS F.
Pulaski, Va.
V. M. D., University of Pennsylvania, 1937. Vouchers: E. T. Booth and J. E. Greer.

WATSON, JAMES HOWARD
607 Olive St., Leavenworth, Kan.
D. V. M., Cornell University, 1938. Vouchers: Col. Ray J. Stanclift and Lt. Col. Geo. J. Rife.

YOUNG, JOSEPH CLIFFORD
Marianna, Ark.
D. V. M., Saint Joseph Veterinary College, 1916. Vouchers: John H. Gillmann and E. B. Mount.

ZICKEFOOSE, EDWARD B.
Rossville, Kan.
D. V. M., Kansas State College, 1939. Vouchers: Edwin J. Frick and R. R. Dykstra.

Second Listing

Cady, Lt. Duane L., Veterinary Station Hospital, Fort Snelling, Minn.

Cain, William, 3328 S. Claiborne Ave., New Orleans, La.

Cochran, Joe Robinson, 533 Tuscaloosa Ave., Birmingham, Ala.

Cook, Raymond W., Pine Road, Fox Chase, Philadelphia, Pa.

Cook, Robert William, 710 Bradley Ave., Peoria, Ill.

Davis, David Everett, 176 Main St., Petaluma, Calif.

Deese, Lester Arvel, Duke Inn, Elizabeth City, N. C.

Eggert, William E., Jr., Office of the Corps Area Veterinarian, Governors Island, N. Y.

Hancock, Edward Erroll Irwin, Provincial Animal Pathologist, Truro, Nova Scotia, Can.

Hanson, John Clarence, 34 W. Central St., Chippewa Falls, Wis.

Harlan, Lt. William Henry 1916 N. W. 38th St., Oklahoma City, Okla.

Hobbs, Joseph William, 1004 Second Ave., Selma, Ala.

Holloway, Albert Lee, Atmore, Ala.

Jenkins, Orville B., 330 Federal Bldg., Madison, Wis.

Jones, Thomas Benton, 2142 W. Van Buren St., Phoenix, Ariz.

Larson, Raymond Edward Towson Veterinary Hospital Towson, Md.

McCarty, Francis A., Rosendale, Wis.

Mainhart, Charles Horine, 350 Woodland Ave., Richmond, Ky.

(Continued on page 544.)

CLINICAL DATA

Streamlined Sheep of China

By SHUPEI SHU, D. V. M., M. S.
Public Health Department, Shanghai Municipal Council, Shanghai, China

SINCE the establishment of the municipal abattoir in the International Settlement of Shanghai in 1893, a number of sheep of both sexes, without horns or ears, have been admitted into the abattoir for slaughter from time to time.

—*After Shupei Shu, 1939.*

Fig. 1. Streamlined sheep of Kiangsu, China. Note the total absence of ear and horn.

These sheep are definitely of a special breed, characterized by the absence of horns and the fact that the external ears are either rudimentary or totally absent. Other features of the anatomy appear similar to normal Chinese sheep, bred for wool and mutton.

This particular type of sheep is bred only in two villages, namely, Li-tang and Bah-mo-tang, at the outskirts of Changshu in the Province of Kiangsu, about 60 miles west of Shanghai and situated in the rich Yangtsze Valley region. Breeders there know nothing of how or when this breed originated. Like many other things Chinese, the fact that this breed is known to have existed in these two villages for many, many years, suffices.

P. Dechambre in *Traite de Zootechnic* stated that the absence or degeneration of the external ear occurs in many species of animals, such as cats, sheep, rabbits and pigs, due to mutation, *viz.*, certain individuals or animals are born without an external ear, this trait being transmitted through subsequent inbreeding to the offsprings. He cited, for example, that in certain villages along the Yangtsze Valley in China, there is a breed of sheep which is characterized by the absence of the external ear, which trait is fixed in this particular breed. These are probably the villages alluded to above. He further stated that in the Department of Aude in France, there is a flock of sheep possessing the same characteristics.

—*After Shupei Shu, 1939.*

Fig. 2. Another view of the streamlined sheep.

Disease in Turkeys Due to Prosthogonimus Macrorchis

By RALPH W. MACY, Ph.D.
College of Saint Thomas, Saint Paul, Minn.

OF THE trematodes parasites of poultry, the most important species in North America appears to be *Prosthogonimus macrorchis* Macy, 1934 (Hegner, *et al*, 1938). It was first reported by Linton, 1887, from a hen's egg from Wisconsin. Kotlan and Chandler, 1925, found that it was the cause of a serious poultry disease at Duck Lake, Mich.

In a controlled experiment undertaken by the writer at the University of Minnesota, in 1934, it was found that chickens infested with these trematodes laid only one-tenth as many eggs as the control group, in spite of few outward symptoms.

Since turkeys are likely to eat dragonflies, which are second intermediate hosts of Prosthogonimus, experiments were conducted to determine the effects of the flukes on these birds.

Two laying turkeys were placed in outdoor pens and fed egg mash and corn to insure proper experimental conditions. On May 13, one was given 347 living cysts of *Pr. macrorchis*, obtained from dragonfly naiads of the genus Tetragoneuria which had been collected from Lake Phalen, Ramsey County, Minn. Six days later this bird was killed and the oviduct was found to contain 35 small but typical *Pr. macrorchis*. The oviduct was nearly in a resting condition. On May 14, the second turkey was fed 20 living Tetragoneuria, which it ate with great relish. Twelve days later 236 medium-sized *Pr. macrorchis* were recovered from the oviduct. Of these, 71 were in the shell gland and the remaining 165 were well distributed throughout the rest of the oviduct. A solid plug of hardened, chalk-gray yolk material, nine inches long, was found clogging a considerable portion of the oviduct. In this mass there were imbedded a number of disintegrating flukes. The body cavity contained large, cream-colored chunks of abortive egg material and much gray pus. Examination of the ovary revealed an apparently normal condition of that organ. There were few external symptoms of disease.

Both of these turkeys ceased laying on the fourth day following the feeding of the cysts. On the previous day each laid a soft-shelled egg.

On June 2, 1936, three additional laying turkeys, kept under similar conditions, were fed Tetragoneuria naiads containing

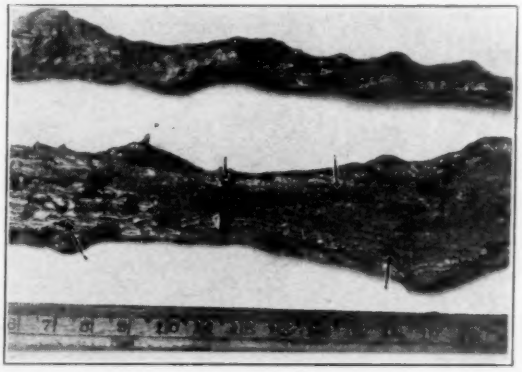


Fig. 1. Lower, opened oviduct of turkey showing trematodes (Prosthogonimus macrorchis), a few of which are indicated by arrows; upper, plug of abortive egg material taken from the oviduct.

an average of twenty-six Prosthogonimus cysts each. The first bird received two naiads; the second and third received eight each. Sixteen days later the turkeys were killed and the first was found to have in the oviduct 52 large *Pr. macrorchis*. The second and third had in the same organ 201 and 208 flukes, respectively. Abortive egg material and pus were found in all of these birds and all ceased laying during the course of the experiment.

While the number of experimental birds was small, the indications clearly pointed to severe disturbance of the genital tract in Prosthogonimus - infected birds. Prostho-

gonimiasis in this case was very similar to that which the writer found in chickens. Turkeys, as well as chickens, are therefore apparently poorly adjusted to this fluke and these birds may be considered as its unnatural hosts.

Turkeys eagerly devour dragonflies, and flocks in the vicinity of lakes may easily become exposed to the infection. Therefore, during the period of maximum dragonfly emergence, May 20 to June 20, the birds should be kept some distance from the lakes.

Fig. 2. Lower, plug of abortive egg material from oviduct; upper, clumps of abortive egg material taken from body cavity.

ACKNOWLEDGMENT

The writer expresses his appreciation to Dr. Wm. A. Riley of the University of Minnesota for laboratory facilities and encouragement in this work.

Bibliography

Hegner, R., Root, F. M., Augustine, D. L., and Huff, C. G.: Parasitology. (Appleton - Century, 1938.)

Kotlan, A., and Chandler, W. L.: A newly recognized disease (prosthogonimiasis) of fowls in the United States. Jour. A.V.M.A., lxvii (1925), n. s. 20 (6), pp. 756-763.

Linton, E.: Notes on a trematode from the white of a newly laid hen's egg. Proc. U. S. Nat'l Mus., x (1887), pp. 367-369.

Macy, R. W.: *Prosthogonimus macrorchis* n. sp., the common oviduct fluke of domestic fowls in the United States. Trans. Amer. Micro. Soc., liii (1934), pp. 30-34.

Macy, R. W.: Studies on the taxonomy, morphology, and biology of *Prosthogonimus macrorchis* Macy, a common oviduct fluke of domestic fowls in North America. Minn. Agr. Exp. Sta. Tech. Bul. 98.

According to an Associated Press report, Ross Hall, a hunter, saw a white-tail deer near Sandpoint, Idaho. He opened fire and thought he saw his target drop. When he hurried to the spot, he found his target—the white tail. The deer was gone.

Use of Iodine for Gapeworms in Pheasants*

By J. P. DELAPLANE, D. V. M., and H. O. STUART

Rhode Island Agricultural Experiment Station, Kingston, R. I.

GAPEWORM infection in pheasants reared in confinement has been a serious problem on some pheasant-rearing farms.

Wehr and Harwood[1] describe the use of barium antimonyl tartrate as a remedy for the removal of gapeworms from chickens. They also give a review of various drugs which have been used for this purpose. Iodine is not among the drugs mentioned, and we should like to cite our experience with this preparation.

A pheasant grower who reared between 800 and 1,000 pheasants in 1938 noted that a number of the birds showed symptoms of respiratory distress arising from severe gapeworm infection, and several of the birds died.

We suggested the use of iodine (iodine vermicide diluted according to the directions for internal medication) to be used by instillation of a few drops in the trachea with a pipette or eyedropper. We also suggested that only the most severe cases be treated, as we were not sure of the safety of the preparation used in this manner.

The owner reported that he had treated 20 severely infected pheasants and that the symptoms of respiratory distress were alleviated after a few days. He stated that he had no further losses from the trouble, and that two birds died following treatment, but from other causes.

Following this experience, two gapeworm-infested pheasants, which were also showing paralytic symptoms, were obtained for treatment and observation. These birds were treated in the manner already described, and the symptoms of respiratory distress disappeared after four and five days. These birds were later autopsied.

*Published with the permission of the Director of Research as Contribution No. 548 of the Rhode Island Agricultural Experiment Station.

Multiple Abscesses in the Skin of Dogs

By W. H. RISER, D. V. M.
Des Moines, Iowa

and E. F. WALLER, D. V. M.
Iowa State College, Ames, Iowa

MULTIPLE abscesses in the skin of puppies caused by *Staphylococcus albus* and characterized by extensive edema is new to us. We have been unable to find reference to the condition in the available veterinary literature. We found a similar condition reported by Fox[1] in man. It is said to occur but rarely in the infant who is ill-nourished, badly fed, and who lives in unhygienic surroundings. The condition has been discussed with small-animal practitioners of considerable experience and they report the condition to be rare.

The condition to be reported occurred in a litter of five Cocker Spaniel puppies. When these pups were two weeks old, the mother developed eclampsia and they were weaned. They were apart from the mother from that date. Five days after weaning, their hind feet began to swell. The lesions were confined largely to the hind legs and did not extend above the hock joints. One of the five animals was not affected. There were marked lesions on the hind feet of the four others and some involvement of the front feet of three of the pups.

The edema was so extensive in the affected portion that at the height of the swelling the skin extended beyond the toe nails. The feet had the appearance of overstuffed boxing-gloves (fig. 1). The hair on all areas that were affected came out and considerable serous exudate, and later pus, accumulated on the swollen skin. The lesions were cultured and the organisms found were identified as *Staphylococcus albus*. We elected to treat these puppies with doses of the toxoid vaccine. The dosage used for each dog was as follows:

10-14-38 .....10 units of toxoid
10-16-38 .....15 units of toxoid
10-19-38 .....25 units of toxoid
10-23-38 .....25 units of toxoid
10-26-38 .....35 units of toxoid

The recovery was complete in all cases of the skin infection. Two of the weakest puppies, however, developed an enteritis on the eighth day of treatment and later died as a result of this disorder. Fox[1] states that gastrointestinal complications are common in the infant suffering with staphylococcic skin infection and that the prognosis is grave. Local treatments of phenyl-mercuric nitrate ointment 1:1,500 and gauze dressings were used. The one pup which did not have the infection and remained with the infected animals, was given no vaccine and remained healthy. The puppies

Fig. 1. Front feet of an affected puppy. Note the boxing-glove-like swelling.

(*Continued from page 538*)

and the examination failed to reveal any gapeworms, indicating that this form of treatment had been effective.

References

[1]Wehr, E. E., and Harwood, P. D.: Barium antimonyl tartrate as a remedy for the removal of gapeworms from chickens. Poultry Sci., xviii, 1, pp. 63-65.

showed no signs of shock or discomfort as the result of toxoid injections.

It is our belief that urine and feces on the skin were the predisposing factors to the infection. This opinion is substantiated by the fact that the infection was more severe on the hind feet than on the front feet. It is also an interesting fact that the infection did not occur until five days after the mother was taken away from the puppies, which would indicate that when the mother could no longer lick and clean the puppies, the skin became unhygienic and infection resulted.

It is also believed that recovery would have been faster if hypertonic solutions of hot magnesium sulfate or saturated solutions of hot boric acid could have been used on the affected parts. Such procedure was almost impossible away from a hospital with so many small puppies.

CLINICAL OBSERVATIONS

Staphylococcic infections of the integument of older dogs are frequently observed in connection with demodectic mange and eczema. It is, however, unusual to observe such a condition in several animals simultaneously. When these pups were submitted, the swollen feet were partially coated with what appeared to be dried serous exudate. This was removed and an attempt was made to sterilize the surface of the affected areas with 70 per cent alcohol. When pressure was applied to the swollen foot, a thin watery pus could be forced from the hair follicles.

A number of smears and cultures were made. Some of the smears were examined fresh for the presence of mange mites, while others were stained. The fresh preparations were negative for parasites but the stained smears revealed staphylococci in large numbers. The cultures produced pure growths of a staphylococcus, later definitely identified as *S. albus*.

In discussing the lesions produced by staphylococci in animals, Minett[2] makes the following classification and statements:

> Infections can be conveniently classed as acute or chronic. Acute changes will include: (a) abscess formation in soft or hard tissue, the lesions being distributed in localized or generalized form; (b) suppurative inflammations of cavities or mucous membranes; (c) gangrene. Under the heading of chronic changes should be mentioned (a) lesions of the usual productive type, and (b) productive lesions in which the type of change is rather more specialized.
>
> When the subject is considered in more detail, there comes to mind the various suppurations of the integument, referred to as furunculosis or as acne, while staphylococci have also been found associated with follicular mange and interdigital abscesses in dogs, and with such essentially necrotic inflammations as fistulous withers, poll-evil and quittor in horses.

In discussing acne in animals, Hutyra, Marek and Manninger[3] state:

> "The staphylococci are probably rarely able, in the absence of other factors, to invade and multiply within the follicles and sebaceous glands. In most cases other favoring circumstances are present, such as mechanical influences, alone or frequently in combination with accumulation of dirt or sweat upon the skin."

The observations made and described here on these four animals are additional evidence that the virulence of *S. albus* can, under certain conditions, be markedly increased.

ACKNOWLEDGMENT

We are grateful to Carl E. Venzke, of the Department of Veterinary Hygiene, Iowa State College, for definitely identifying the microörganism for us. The clinical observations were made by W. H. Riser and laboratory observations by E. F. Waller.

References

[1]Fox, C.: Taken from "Diseases of the Skin," by Ormsby. (4th ed., 1934), p. 333.

[2]Minett, F. C.: Staphlococcus infections in animals, with special reference to toxoid in preventive and curative treatment. Vet. Rec., xlix (1937), pp. 179-186.

[3]Hutyra, Marek, and Manninger: Pathology and Therapeutics of the Diseases of Domestic Animals. (4th English ed., iii), p. 567.

Attorneys for Mrs. Mabel Jones West, of Birmingham, Ala., paid $280 in Washington, D. C., to cover printing and other costs of an appeal to the Supreme Court against a Birmingham ordinance requiring her to have her Pekingese inoculated against rabies.—*Time.*

Nitrate as the Cause of Oat Hay Poisoning*

By W. B. BRADLEY, D. V. M.,
H. F. EPPSON and O. A. BEATH
Wyoming Experiment Station, Laramie, Wyo.

IN A PREVIOUS paper (in press) we have shown that animals poisoned by the ingestion of oat hay or oat straw die from methemoglobinemia. Work with water extracts showed the toxic principle to be water soluble and heat stable.

Concentration of these extracts caused the separation of large amounts of crystalline material, which was found to be potassium nitrate. The amount of KNO_3 in samples of oat hays and straws from ranches where live stock losses have occurred varied from 3.2 to 7.2 per cent. The nitrate seems to occur mainly in the straw, for analyses gave 3.5 per cent KNO_3 in hay, 0.7 per cent in chaff and only a trace in the grain.

To prove that the potassium nitrate in the oat hays and water extracts of these hays is the lethal factor, 15 pounds of oat hay was extracted twice with boiling water, making a total extract of seven gallons. Two gallons of this extract containing 128 grams of KNO_3 was given by stomach tube to a 255-pound calf, which showed progressively higher concentrations of methemoglobin until its death about 10 hours later. In blood drawn after death it was found that about 65 per cent of the hemoglobin had been converted into methemoglobin. At the same time another calf weighing 277 pounds was given an equivalent quantity (141 grams) of KNO_3 dissolved in 2 gallons of water. Symptoms and methemoglobin formation closely paralleled those of the other animal. Death occurred in seven hours, with about 80 per cent of the hemoglobin converted into methemoglobin. To prove further that potassium nitrate is the toxic factor, two gallons of the same oat-hay extract, from which about 70 per cent of the KNO_3 had been removed by crystallization, was given by stomach tube to a 260-pound calf without any ill effect.

We have been unable to obtain any evidence that oat hay contains any nitrite. However, water extracts of oat hay slowly develop a small and probably insignificant amount of nitrite. Since the urine and bile of poisoned animals contain nitrite, and since nitrite is necessary for the formation of methemoglobin, this conversion of nitrate to nitrite must occur in the gastro-intestinal tract.

Although sheep have been reported[1, 2] as being unaffected by oat hay, a ewe weighing 140 pounds was fed 2.2 pounds of ground oat hay by stomach tube. This animal developed some methemoglobin and some respiratory distress and weakness but recovered. Four days later this same ewe was fed 2.2 pounds of the same oat hay. After this feeding no methemoglobin could be found. The animal died during the night and, at autopsy, there was no methemoglobin in the blood.

Another ewe weighing 107 pounds was given two gallons of water extract from four pounds of oat hay. This extract contained 107 grams of KNO_3. Blood drawn before death and at autopsy showed small quantities of methemoglobin. Because of the small amount of methemoglobin formed, it would appear that sheep are less capable of converting nitrate to nitrite than cattle and therefore die from nitrate rather than nitrite poisoning.

Perhaps KNO_3 is the toxic factor in many plants which, although ordinarily harmless, sometimes cause rapid death when fed. We have analyzed one sample of pigweed which was reported to have killed several calves and found this weed to contain 3.6 per cent KNO_3, a concentration which is well within the limits found in the toxic oat hays analyzed.

Further work is in progress to determine what factors are responsible for the development of such high concentrations of

*Published with the approval of the Director of the Wyoming Experiment Station.

Calcium Therapy*

By L. N. MORIN, D. V. M.
Clinton, Ill.

THE INTRAVENOUS use of calcium preparations has become so widespread for a variety of conditions that its popularity, if not directed into the right channels, may act as a boomerang, and these preparations may meet the same fate of many good products which have suddenly become publicized. The purpose of this study, therefore, is to attempt to evaluate the use of calcium, in its several combinations, in the treatment of deficiency diseases such as milk fever, ketosis and other conditions presenting a similar clinical picture.

Our observations over a nine-year period, during which we have made approximately 3,000 intravenous injections, lead us to believe that the most important point in the intelligent use of calcium products is the recognition of the fact that the response following their use is, after all, only transitory. Steps always should be taken to give supporting after-treatment.

Dosage, both as to kind and amount, is in our opinion a question of response following treatment in each individual and the determination of the presence or absence of ketosis by use of the urine test. Our records show a number of cases of ketosis in cows, during the grass season, that responded well to calcium gluconate and increased dosage of dextrose and, later at the calving period, showed the same symptoms, excepting that the ketone test was negative. In cases of this type it is certainly plain that the question of what and how much to give is a very important matter and should not be dismissed by mere routine injection of some form of calcium preparation.

We must not forget that the presence of ketone bodies in the urine tells us of the need for sugars but does not indicate anything as to the calcium level. Until we have a field method of testing for calcium, we favor the use of calcium along with dextrose in known ketone cases. Furthermore, calcium has a valuably stimulating effect. In fact, it appears that this stimulation is one of the principal points in its effectiveness.

Since we began to conduct the urine test on all cases, we have detected definite ketone reactions in approximately 20 per cent of these, while the relapses associated with both types during the same period proved to be 12 per cent of all cases, equally divided as to ketone reactors and non-reactors. The more we study along this line, the more we are convinced that dextrose is a valuable adjunct to calcium preparations and, where ketosis is present, should be used in increased amounts. The size of the cow, her condition as to flesh, how heavy a milker she is, how rugged a calf she may have dropped, or how near death she may be when we see her, are things that should be carefully considered.

The intravenous method of treating milk fever, with the ensuing spectacular results, has given our profession much favorable publicity. But occasional sudden death during injection has brought publicity of the wrong kind, and promoted caution on the part of the veterinarian.

We believe that intravenous medication merits wider use, but the pitfalls accompanying this direct form of treatment call for more careful technic.

The picture, as we see it, in an extreme case of milk fever, is of a heart that is at such a low ebb that circulation has almost

*Presented at the fifty-seventh annual meeting of the Illinois Veterinary Medical Association, Springfield, February 16-17, 1939.

(*Continued from page 539*)

KNO_3 in oat hays grown on certain areas.

References

[1]Newson, I. E., Stout, E. N., Thorp, Frank Jr., Barber, C. W., and Groth, A. H.: Oat hay poisoning. Jour. A. V. M. A., xc (1937), n. s. 43 (1) pp. 66-75.

[2]Thorp, Frank Jr.: Further observations on oat hay poisoning. Jour. A. V. M. A., xcii (1938), n. s. 45 (2), pp. 159-170.

ceased—therefore, a very sluggish blood stream containing a minimum of oxygen. Such a condition calls for very slow injection, by the gravity method only, especially when one is using material that has such prompt action as calcium salts possesses. We are not satisfied with our treatment in these cows unless we see a return of nervous sensation, evidenced by trembling, prompt bowel evacuation and ability to arise in a short time. If the above effects are absent or greatly delayed, we feel certain that the dosage has been insufficient or that our diagnosis was faulty. Soon after injection, and where the response has been satisfactory, turning the cow to her other side and touching with an electric stock gad creates a lasting impression on both patient and owner, and replaces punching and kicking often resorted to in making the animal rise.

Inasmuch as we consider the patient's ability to come to her feet as a gauge to proper dosage, we invariably stay on the job until she stands or until we have discovered some complication that prevents her from standing.

The use of calcium products, in our hands, has evolved from home made calcium chloride solution, 10 per cent, in 125 cc. doses, to our more frequent dosage at this time of 250 cc. calcium gluconate, 23 per cent, and 250 cc. dextrose, 50 per cent.

We have experienced that unfortunate accident of calcium chloride sloughs, as well as those all too frequent relapses. The former are now definitely a thing of the tragic past. While there are still occasional relapses, the fact that they are becoming more rare is, we believe, an indication of more careful diagnosis and dosage.

Sloughing of the skin is not pathognomonic in swine erysipelas. It can occur also in "suipestifer infection." . . . Calcium-deficient pigs are more susceptible to swine erysipelas . . . paint-brush redness of viscera is seen in other infectious diseases.—*J. D. Ray, before the Iowa Veterinary Medical Association.*

An Ictero - Hemoglobinuria - Like Disease in Florida

By M. W. EMMEL, D. V. M., and D. A. SANDERS, D. V. M.
Florida Agricultural Experiment Station, Gainesville, Fla.

BACILLARY ictero-hemoglobinuria, commonly called "red water disease" and "hemorrhagic disease," which principally affects cattle but rarely sheep, was first reported as occurring in cattle in California, in 1916. The disease occurs during the summer and early fall in swampy, poorly drained areas and is known to exist in the mountain valleys of the Sierra Nevada and coast ranges in the United States and the Andes in southern South America.

Bacillary ictero-hemoglobinuria is characterized by sudden onset, rapid course, high temperature, hemoglobinuria, from which the term "red water" was derived, and occasional bowel hemorrhage.

Postmortem examination reveals bloody discharges from body openings, hemorrhages on the mucous membranes, subcutis and serous surfaces of the viscera and pleura, and hemorrhagic infarcts on the liver. Hemoglobinuria is constantly observed. The blood is watery and hemolyzed, the hemoglobin reading is lowered, and the red blood-cell count is reduced seriously during the course of the disease.

Vawter and Records in 1922 isolated an anaerobic, spore-forming organism from liver infarcts of animals affected with this disease. Ictero-hemoglobinuria has been induced experimentally in cattle with this organism, which is also pathogenic for rabbits and guinea pigs. Pathogenicity depends upon an unstable hemolytic toxin. These investigators perfected a vaccine, a killed, undiluted broth culture of the causal microörganism, as a prophylactic and an antiserum for the treatment of the disease.

During the present year, from April until the middle of July, eight animals, two to seven months of age, and one cow 13 years of age, in the University dairy herd became affected with a disease bearing a close resemblance to bacillary ictero-hemo-

globinuria. The only deviation from the known lesions of this disease appeared to be the absence of the liver infarct which Vawter and Records report to be a constant lesion. Only one animal showed this lesion. Hemoglobinuria was observed in three animals seven days prior to death and was present in all animals at death. The blood was watery and hemolyzed, the visceral organs icteric and usually hemorrhagic. The peritoneal and pleural cavities contained bloody fluid.

After a tentative diagnosis of ictero-hemoglobinuria was made, 60 lactating cows and 60 nonlactating animals were vaccinated with ictero-hemoglobinuria vaccine. During the third week following vaccination, four abortions occurred among the lactating animals. The embryos ranged from two to approximately seven months of age. The outstanding lesions shown were hemolytic in character. The hypothesis was advanced that the hemolytic toxin of the vaccine was responsible for these abortions in animals, which were probably not in the best of physical condition. However, Records states that storage of the vaccine results in the formation of non-toxic toxoids. Since vaccination, no new cases of the disease have developed in the dairy herd.

Although the livers of three autopsied animals were cultured, the anaerobic organism, *Clostridium hemolyticus bovis*, was not isolated. Consequently there is some question as to whether or not the disease was true bacillary ictero-hemoglobinuria. Records and Vawter, after being informed of the history of this outbreak, are of the opinion that the disease was not bacillary ictero-hemoglobinuria.

More recently, however, a purebred Hereford bull in the University beef herd developed hemoglobinuria. This herd had not been vaccinated. The animal had been indisposed for four days before hemoglobinuria was discovered. The urine was the color of venous blood. The hemoglobin reading was 15 as compared with a normal of approximately 80-90. The temperature was 104°. As soon as hemoglobinuria was observed, the animal was given 400 cc. of hemoglobinuria antiserum intravenously, after which there was progressive improvement. Hemoglobinuria disappeared within 48 hours. A second injection of antiserum was administered intravenously on the third day. Recovery now appears to be complete. A communication from Records states that the history of this case and the immediate response of this animal to the specific hemoglobinuria antiserum would tend to indicate that perhaps the disease is true bacillary ictero-hemoglobinuria.

According to reports, this is the first time a disease of this type has been reported in Florida. In case the disease is bacillary ictero-hemoglobinuria, it seems logical that it will appear elsewhere in the state. This report is being made in the hope that it will be of value in the recognition of the disease and in the determination of its true character.

Applications

(*Continued from page* 535)

Maurer, E. Laverne, 304 Federal Bldg., Great Falls, Mont.

Nagle, Albert C., Box 174, Harrisburg, Pa.

Reichert, Paul F., Route 2, Chelsea, Mich.

Ritter, Earl Conrad, Sumner, Iowa.

Safford, John W., Box 129, Lewiston, Idaho.

Schneider, Morris D., Box 154, Boscobel, Wis.

Schwartz, Albert V., Jr., 326 Post Office Bldg., Baton Rouge, La.

Scrivner, Lloyd Herbert, Cornell University, Ithaca, N. Y.

Shelby, Clarence Franklin, 326 Post Office Bldg., Baton Rouge, La.

Strandberg, H. L., Glenwood, Minn.

Terry, Thomas A., Calle A esquina a 21, Vedado, Havana, Cuba.

Theophilus, Donald K., Fort McPherson, Ga.

Tracey, Richard W., Parkton, Md.

Van Sant, Willard Merrill, 824 E. State St., Boise, Idaho.

Weisner, Ernest Steven, 1009 W. Grand River Ave., East Lansing Mich.

Whitehead, Lt. Charles Joseph, 2711 S. 13th St., Tacoma, Wash.

Wilkin, William Arthur, 413 S. Sycamore St., Centralia, Ill.

Ziebell, Vernon Frank, Tomah, Wis.

Zinober, Moses Robert, 602 Lincoln St., Antigo, Wis.

The amount which should accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1940, including subscription to the JOURNAL.

Nonsurgical Relief of Intussusception

By S. W. HAIGLER, D. V. M.
Saint Louis, Mo.

THE PATIENT, a Cocker Spaniel, male, aged five years, was presented at the hospital on October 7, 1938. The owner explained that the dog had been out for about two hours and, upon returning home, seemed greatly distressed. The abdominal cavity was distended and very shortly the animal vomited large quantities of garbage, after which he seemed comfortable again, the owner stated. Within the next hour he had several convulsions and was brought to the hospital immediately.

Examination revealed a slight tenseness of the abdominal cavity. The pulse and respiration were slightly above normal; the general appearance was near normal. One-half ounce of milk of magnesia was given and a digestive tablet dispensed. Then the patient was taken home.

On October 9, the dog was returned to the hospital. The owner reported that the appetite was good but that there was considerable vomiting. The pulse was slightly accelerated, and the temperature registered 103°. A mild laxative and an enema were given. The patient remained under care until October 11, at which time he was discharged—apparently normal.

From October 11 to October 20, when the animal was readmitted to the hospital, the owner reported the dog in fair condition, except for intermittent periods of diarrhea. In our examination of October 20 a lump was detected by palpation in the abdominal cavity. Fluoroscopic and x-ray examination revealed an enlargement in the jejunum and gas distention of the bowel anterior to it. A diagnosis of intussusception was made. Since the patient was very weak at this time, an operation seemed inadvisable.

A proprietary extract was given in 2 cc. doses intramuscularly every four hours, four times. In addition 200 cc. of 5 per cent dextrose in Ringer's solution was administered at twelve-hour intervals.

On October 21, the patient seemed much improved. On palpation there was considerably less soreness of the abdominal cavity. Four additional doses of tissue extract, 2 cc. each, were given at four-hour intervals. The dextrose treatment also was continued.

The following day, the patient appeared quite lively, and took and retained broth and water. Upon palpation, the enlargement in the intestine that was noted on the two previous days could not be detected. It was also negative upon fluoroscopic examination.

There was slight improvement each day for the next three days. But on October 26 there was marked depression and the respiration and pulse were accelerated. The temperature was 105°. The administration of dextrose was continued and a saline enema was given. Routine treatments during the next four days produced no improvement. The patient died on October 31.

Autopsy findings: Small quantity of fluid in the abdominal cavity; stomach entirely empty; extensive inflammation and gas in duodenal and upper small intestine; great distention of ileum and colon, four times normal circumference, partly filled with gas-forming fecal material; mucous membranes partly absent from ileum and colon. There was a very pronounced circular band in the region of the intussusception, and a marked degree of peritonitis was present.

Conclusion: I am definitely of the opinion that the intussusception was relieved after the first few injections of tissue extract. Whether the great distention and thinness of the walls of the ileum and colon were due to repeated injections or to the long duration of the condition, I can not be certain, but I am inclined to attribute this to the latter.

LEGAL OPINIONS

By JOSEPH M. KOTZ
Attorney for the Association

Special Contract

As a general rule, the veterinarian can diminish his liability by special contract, the principle being that he may impose whatever terms he chooses. If the owner is given notice of the special provisions affecting the transaction and if he agrees to deliver the animal under these circumstances, he is bound by them, provided that the contract is not in violation of the law or of public policy and that it stops short of protection in case of fraud or negligence of the veterinarian.

Cases arising from misunderstanding or lack of information concerning special terms usually involve certain printed forms of contracts or special receipts, wherein in fine print there are such expressions as "at owner's risk," "not responsible for any loss," or "not responsible for loss by damage, fire, flood, or other agencies, unless caused by wilful act or gross negligence." In such instances, the veterinarian's liability or immunity from liability must be decided.

In the case of the *X Co.* vs. *S*——, 169 Pac. 262, the court said:

> The writers on bailments (the legal title under which this subject is discussed) seem to agree that the parties to a bailment contract may regulate the responsibilities of the bailee by special contract, but it is also universally agreed that the terms which public policy and legislation of the state impose are not to be overleaped by contractual relations and, if so, the contract will be disregarded and declared void, and the bailee held in the same manner and to the same extent as if such contract never existed * * *.

It is the better rule and generally upheld in our courts that a bailee (veterinarian) for hire can not, by contract, so limit his responsibility to the bailor (owner of animal) as not to be liable for his own negligence or the negligence of his agents and servants.

The Ohio Supreme Court has held:

> As a bailee for hire impliedly contracts to use ordinary care, it follows that he may not contract against his own negligence or the lack of such care. Ordinarily, in bailments the parties may diminish the liability of the bailee by special contract, provided the contract is not in violation of law or of public policy and does not relieve the bailee of negligence.

In *R*—— vs. *F*——, 106 S. E. 303, the court held that an auto repair man, being a bailee for mutual benefit, could not limit his statutory duty to exercise ordinary care by posting a notice in his garage to the effect that he would not be responsible for losses due to fire or theft. It appeared that the plaintiff had left his automobile in the defendant's garage for repairs. The plaintiff had posted a notice denying responsibility for the loss of articles by fire or theft. The Georgia statute provided that the bailee was bound to exercise ordinary care to protect property held under a mutual benefit bailment. While the automobile was in the garage, it was stolen by one of the defendant's employés.

A veterinarian, however, may limit the extent of his liability to the owner of an animal, granted that this limitation is not unreasonable. But there have been cases where such limitations on liability have not been upheld, simply because they seemed unreasonable.

South Carolina forbids dental operations on horses or mules to conceal their age.

In Boston, Mass., it is unlawful to keep a dog more than ten inches in height.

A city law at Topeka limits any household to the maximum possession of five cats.

CURRENT LITERATURE

ABSTRACTS

AVIAN BRUCELLOSIS. (Title translated.) P. Pavlov. Rec. de Méd. Vét., cxiv (1938), pp. 790-798.

Because brucellosis is an important disease, it has attracted the attention of numerous scientists. Although widely investigated in mammals in all countries, its study in fowls has been somewhat neglected, notwithstanding that it has been known to affect chickens since Dubois in France (1910) signalized a grave epizoötic of avian brucellosis of bovine origin, and the researches of Emmel and Huddleson in America (1928-30) proved that the disease exists in farm fowl. The agglutination titre may remain high for 1½ years after infection, but the bacteria disappear from the blood rapidly. It is not transmitted by eggs, and young fowl are quite resistant.

Chickens artificially infected by intraperitoneal, intramuscular, and intravenous injections showed an agglutination titre of 1:320 on the fourth day and of 1:10,000 on the tenth day. Out of 32 birds inoculated, cultures were isolated from twelve, of which six had been injected intramuscularly, five intravenously and one intraperitoneally.

Thomsen, in Denmark, who conducted feeding experiments on 2,677 chickens, was not able to produce the disease *via* the digestive tract, except in 0.97 per cent; and in this small number the titre was only 1:20 to 1:50. The conclusion was that avian brucellosis is of no particular significance in Denmark.

Pigeons, however, showed a higher degree of susceptibility to *Brucella melitensis* of human origin; but, in the work of Pavlov, the receptivity of chickens to *Br. abortus* was greater than that of pigeons.

Summarizing his experiments, the author declares that domestic fowls are much more difficult to infect than mammals, by either natural or artificial methods; the agglutination reaction reaches its maximum a month after infection; the agglutinins disappear in two months; eggs artificially infected *en masse* retain the organism for only four to 14 days; and that the disease in chickens has no clinical symptoms.

PATHOLOGICAL ANATOMY OF EXPERIMENTAL THROMBOPENIC PURPURA IN THE DOG. Leandro M. Tocantins and Harold L. Stewart. Amer. Jour. Path., xv (1939), p. 1.

Clinical and pathological manifestations of experimental purpura as produced with antiplatelet serum in the dog may be divided into the following stages: (1) acute stage (1st to 5th day), exhibiting thrombopenia, prolonged bleeding time and, in the tissues, hemorrhage, edema and pigment deposition; (2) intermediate stage (5th to 10th day), during which there is a rising platelet count, short bleeding time and multiple vascular thrombi in various organs, principally in the spleen; (3) reactive stage (after 10th day) characterized by high blood-platelet count and hyperplastic changes in the bone marrow, spleen, lymph nodes, thymus and Peyers patches of the ileum.

The intensity and distribution of the hemorrhages seem to be largely conditioned by the trauma and the degree of internal and external stress normally undergone by the part. Congestion and partial obstruction in the circulation of lymph are evident during the acute phase of purpura. Lymph vessels and sinuses of lymph nodes draining hemorrhagic areas become greatly distended with blood. Edema and failure of hemorrhage resorption may be the result of failure of the lymphatic circulation.

A COMPARATIVE STUDY OF DISTEMPER INCLUSIONS. R. G. Green and C. A. Evans. Amer. Jour. Hyg., xxix (1939), p. 73.

The specific inclusions of epizoötic fox encephalitis are intranuclear and occur most often in vascular endothelium, but they also are found in cells of the reticuloendothelium, hepatic cord cells and occasionally elsewhere in foxes and dogs. The inclusion bodies of canine distemper are both principally in epithelium lining various passageways, such as the urinary tract, the respiratory system, the bile ducts and the ducts of the salivary glands and the pancreas, but also are found in certain cells of the adrenal gland, lymph nodes, spleen and other organs.

Distemper inclusions do not occur in cells of the vascular endothelium and are present only rarely in the hepatic cord cells. Canine distemper and epizoötic fox encephalitis can be differentiated with certainty by their respective inclusion bodies. Although the inclusion bodies of canine distemper in dogs, foxes, minks and ferrets show minor differences, they appear morphologically identical in all four species.

THE WOLF TOOTH OF SOLIPEDS. (Title translated.) Marcel Petit. Rev. de Méd. Vét., xc (1938), pp. 619-628.

The name "wolf tooth" is given to the rudimentary pre-molar of the soliped because, morphologically, it resembles the canine tooth, if not also because of its location on the bars. It was first described by Daubenton in 1763 and was studied by Goubaux and Barrier in 1890 and by Lesbre in 1892. However, numerous points about its morphology and evolution remain to be elucidated.

Wolf teeth occur in both jaws (maxillary and mandibular) more often, however, on the upper jaw. The lower ones are less developed and may remain hidden under the mucosa; they are pointed, delicate stylets exposed to bit injury which those of the upper jaw escape.

This rudimentary tooth is molar, not canine in origin. It is found to have been well developed in the "ancestors of the horse," the *Miohippus brachylophus* of the American Miocene period and the *Parahippus crenidens* of the middle Miocene. Progressively, the wolf tooth declined to a simpler form through the species of successive eras, never, however, to disappear entirely. Always it was more developed in the upper arcades and, at the present time, specimens possessing the true picture of a maxillary molar are still found.

Wolf teeth are more frequently found in horses between the ages of six and ten years and, strangely, they are more common in geldings than in stallions or mares. The ratio was 10 per cent for stallions, 21 per cent for mares and 24 per cent for gelding, in an extensive observation made on military horses of the Arabian breed. They are somewhat more frequent on the right than the left side. The difference was 91 for the right side and 73 for the left in 144 horses examined. Paleontologists and anatomists are not in accord as to whether wolf teeth belong to the temporary or permanent dentures. Some authors contend that they are temporary teeth because they develop with the deciduous dentition and precede the appearance of permanent replacements. According to others, they should be classified with the permanent teeth. All agree, however, that this unit of the dental mechanism is gradually degenerating and disappearing, a fact that stands out in bold contrast with the outstanding improvement of the premolars of horses as time goes on. "These have become larger and more complicated than the molars and have transformed the tables of the dentures of solipeds into a machine remarkably arranged for grinding forage."

VARIATIONS IN THE NUMBER OF VERTEBRAE OF SWINE. V. A. Freeman. Jour. of Heredity, xxx (1939), p. 61.

Considerable variation exists in the number of ribs, thoracic vertebrae and lumbar vertebrae of pigs. Some strains of certain breeds inherit, on the average, larger numbers of vertebrae than others, but the variation existed in the five breeds examined

and was about the same in each sex. The number of ribs appear to be influenced by more than a single pair of genes. Carcasses with 21 and 22 body vertebrae were longer than carcasses of similar breeding with 20 body vertebrae. Selection for length of body is believed to have resulted in some increase in the number of body vertebrae.

THE INFLUENCE OF VITAMIN A UPON UREA AND INULIN CLEARANCE IN THE DOG. Raymond C. Herrin and Henry J. Nicholas. Amer. Jour. Physiol., cxxv (1939), p. 786.

Diets containing 150 grams of butter or cod liver oil increased the postabsorptive urea clearance of dogs 30 to 47 per cent, respectively. Later the clearance with the cod liver oil diet fell below normal with the presence of red and white cells and casts in the urine. A diet containing 150 grams of lard, supplemented with the vitamin D equivalent of the cod liver oil, resulted in no increase in clearance and a final decrease of 42 per cent. Such a diet supplemented with the vitamin D and the vitamin A equivalent of the cod liver oil diet resulted in a 60 per cent increase in the urea clearance. Supplementing the diet presumably containing sufficient vitamin A with 50,000 units of vitamin daily in the form of halibut liver oil resulted in a 41 to 94 per cent increase in the urea clearance. The earliest increase occurred within a week; the maximum increase occurred after 96 days.

In one dog the clearance remained elevated for 44 days after the supplement was withdrawn. In a dog which had been maintained on a deficient diet for five months prior to conception, the urea clearance decreased 40 per cent during pregnancy; parturition was normal, but lactation was a failure. Carotene increased the clearance 34 per cent above normal. Vitamin A deficiency was produced in twelve young and three adult dogs. In all of these dogs the clearance decreased but rose to above normal when the diet was supplemented with 10,000 to 50,000 units of vitamin A daily. The clearance cannot be kept permanently elevated with vitamin A.

EIN FALL VON THROMBOSE. (A case of thrombosis in a gelding.) Wurfschmidt. Zeit. f. Veterinärk., li (1939), p. 25.

Treatment of severe cases of thrombosis is regarded, usually, as vain. In the case described by Wurfschmidt an intravenous injection of "fibrolysin" (15 per cent aqueous solution of thiosinamin) was used, repeating the doses six times at intervals of two days. Two weeks later, gradual exercises were prescribed. After four weeks, Sklerostomex, a proprietary vermifuge, was given intravenously, and the doses repeated eight times at seven-day intervals. Ten days after the first injection of Sklerostomex, the horse was able to trot for ten minutes; two weeks later it was able to trot for 35 minutes, and after seven weeks it could trot for 55 minutes without showing symptoms of fatigue. At the end of six weeks the horse appeared to have recovered entirely.

FURTHER OBSERVATIONS ON FILTRABLE TUMORS INDUCED IN FOWLS BY INJECTION OF TAR. James McIntosh and F. R. Selbie. Brit. Jour. Exp. Path., xx (1939), p. 49.

Four tar-induced tumors were propagated in series and, of these, two have been transmitted by Berkefeld filtrates. One of the nonfiltrable tumors was passed by means of emulsions and the other by means of minced tissues only. Another tumor, not transmissible by Berkefeld filtrates, was transmitted by paper-pulp and sand filtrates.

The authors conclude that the transmission of these tumors is indicative of an active virus. Variations in transmission are considered to be due to variation in the relation of the virus to the tumor cells, to a decrease in virulence, to the presence of an inhibitory substance in the filtrate, or to the virus being too large to pass through the Berkefeld filter. It is considered that tar-induced sarcomas of the fowl are virus tumors and, therefore, presumably filtrable. Carcinogenic agents induce the tumors but play no part in the maintenance of the subsequent malignant process.

THE INCIDENCE OF UNDULANT FEVER. Jour. Lab. & Clin. Med., xxiii (June, 1938), pp. 218-221.

The occurrence of undulant fever in man computed from positive immunological reaction in 5,000 blood-serum examinations made among the patients of a general hospital was 51 agglutinations in a dilution of 1:25 and nine in dilutions of 1:25 to 1:100. That is to say, 60 patients out of 5,000 had positive reactions. In the follow-up study of these reactors it was revealed that, in 39 of the group, 24, or 62 per cent were living on farms and were habitual drinkers of raw milk during the preceding two years. Eight of them, however, had drunk little or no raw milk.

In the series of 5,000 blood-serum samples tested, 38 per cent were from men and 62 per cent from women. Of the 60 positive serums, 35 per cent were from men and 65 per cent from women. The incidence in men and women was therefore about equal.

Studied as a whole, this observation sets the percentage of positive immunological reactions in an unselected group at 1.2 per cent, shows that sex is not a factor, and that there is a relationship between undulant fever reactions and the ingestion of raw milk.

NEPHRITIS AND ITS INFLUENCE UPON HEMOGLOBIN PRODUCTION IN EXPERIMENTAL ANEMIA. G. H. Whipple and F. S. Robscheit-Robbins. Jour. Exp. Med., lxix (1939), p. 485.

There has been an 11 per cent incidence of glomerulo-nephritis in the authors' anemic dog colony. The course of nephritis is insidious, usually extending over several years and ending in uremia, often with terminal bronchopneumonia. Hemoglobin formation in these standard anemic dogs is well established as related to various standard food factors. Nephritis causes little or no change in hemoglobin production in anemic dogs in the early stages of the disease. In the later stages of nephritis there may be no change or moderate changes in hemoglobin production in these anemic dogs. The average is 70 per cent of normal hemoglobin production in advanced nephritis. It seems unlikely that this degree of impairment of hemoglobin production in nephritis would result in spontaneous anemia in the dog.

THE ANTIBODY RESPONSE TO SWINE INFLUENZA. Carlos T. Rosenbusch and Richard E. Shope. Jour. Exp. Med., lxix (1939), p. 499.

Antibodies that neutralize swine influenza become detectable in the serum of swine on the sixth or seventh day after infection with swine influenza. Their appearance corresponded closely with clinical recovery. In swine with the milder filtrate disease-neutralizing antibodies did not appear until sometime between the seventh and tenth days. The maximum antibody titres ranged from 1:60 to 1:160 and were attained on the 14th to the 27th day after injection. There was no apparent relationship between clinical severity of disease and the maximum antibody titres eventually reached. It seems apparent that the appearance of the circulating, virus-neutralizing antibody is not the sole cause of clinical recovery.

THE USE OF SULFANILAMIDE IN EXPERIMENTAL BRUCELLOSIS. Ben D. Chinn. Jour. Inf. Dis., lxiv (1939), p. 78.

Sulfanilamide has a bactericidal and bacteriostatic effect on *Brucella melitensis, B. abortus* and *B. suis* in vitro. The drug has been found to have a protective action in all guinea pigs inoculated with these organisms when treatment was begun immediately after inoculation. Sulfanilamide has a definite therapeutic effect in Brucella-infected guinea pigs when treatment is begun one week after inoculation. From 50 to 100 per cent of the animals, depending on the type of organism inoculated, were negative pathologically and culturally after treatment, while untreated animals were all positive for Brucella and showed characteristic lesions. Infections in guinea pigs

due to *Br. abortus* and *Br. suis* responded more satisfactorily to sulfanilamide treatment than infections due to *Br. melitensis*.

CASTRATION OF PIGS AT VACCINATION TIME. L. N. Morin, Veterinary Medicine, xxxiv (May, 1939), p. 305.

The castration of pigs when they are given serum and virus in vaccinating against cholera has absolutely no harmful effect and has the advantage of being a time-saving practice. Inasmuch as the serum is injected into the peritoneum, the position is ideal for castration. The testicles fall forward and leave the scrotum loose and empty. The scrotal skin is lifted up with the left hand and incised on each side with a hook-blade knife. Tipped out of the incision with the fore finger, the two testicles are pulled upon strongly and boldly severed at the surface of the body, whereupon the stump retreats into the abdominal cavity sheltered from infection.

THE EFFECT OF SULFANILAMIDE ON EXPERIMENTAL INFECTIONS WITH BACTERIUM NECROPHORUM IN RABBITS, Elizabeth S. Hemmens and G. M. Dach. Jour. Inf. Dis., lxiv (1939), p. 43.

Sulfanilamide has given good results in the treatment of rapidly fatal infections with *Bacterium necrophorum* in rabbits. Maximum effect was obtained when the treatment was started by the third day of the infection or earlier and continued until the lesion had regressed. The effect of the drug was to stop the spread of the necrotic process.

Histologically, there was a connective tissue wall infiltrated with heterophil leukocytes in the animal treated for 4 days or longer. This process eventually resulted in complete encapsulation if treatment was continued. There were fewer bacteria in the abscess of the treated as compared with the untreated animal. The mode of spread of the infection seemed to be through the blood vessels.

BOOK NOTICES

EMOTIONS AND BODILY CHANGES. H. Flanders Dunbar, M. D., Med. Sc. D., Ph.D., Department of Medicine and Psychiatry, Columbia University. 2nd edition. 600 pages. Columbia University Press, New York City, 1938. Price $5.00.

That the relationship of emotions to disease is not a conjecture in human medicine is evidenced by the publication of a second edition on a subject which, in animal medicine, would seem quite vague but which is as much a part of human pathology as other etiological factors. In other words, there is a psychosomatic unity governing function, dysfunction, and actual morbidity of an organic as well as of a general nature which is not to be overlooked in the practice of medicine. As the author states in his introduction, the "hook up" not only marks a new era in medicine but is a stumbling block in scientific research and in the study of personality, which now should be "gathered together, correlated and evaluated." As a matter of fact, *psychosomatic medicine* is no longer to be set apart from the usual specialties. It belongs within, not outside of, medical doctrines, because among other logical reasons, the psychic etiology of illness is an established fact.

The book is divided into four parts, namely: Orientation and Methodology; Organs and Organ-Systems, Therapeutic Considerations, and a bibliography of 134 pages, where the thirst for details may be satisfied.

A reading of this fascinating volume raises the question of psychosomatic interrelationships in animal pathology, where instinct replaces reason and where the emotions have yet to be included in our studies of pathology and therapeutics. Certainly such emotions as fear, disappointment, excitement, and pleasure do play a definite rôle in diseases of animals, because in the absence of reason these are more highly intensified than in the human, who can modulate such emotions by using the common sense with which animals are not endowed.

In reviewing this book one is prompted

to say that if the author had been a long-time practitioner of veterinary medicine whose sole guide in diagnostics are objective phenomena, he would have had considerable advantage in computing the influence of such emotions as fear, anger, joy, etc., on the course of morbid process and the reflexes which govern the normal functions.

We recommend the book to the "book worm veterinarian" who craves for knowledge beyond the usual drift.

MEDICAL OCCUPATIONS. Lee M. Klinefelter. 285 pages, with 67 graphic illustrations. E. P. Dutton & Co., Inc., New York City, 1938.

An informative book—the first of its kind—describing the various branches of medicine a young man may choose as a career: human medicine, veterinary medicine, dentistry, pharmacy, chiropody, optometry, physical therapy, medical technology, nursing, x-ray technics, government medical service.

The book covers the educational requirements, length of courses, licensure, graduate work, prospects and, especially, the daily grind of those engaged in the practice of these several occupations. It also contains lists of the colleges the young man may select for his alma mater, and cites the literature he may consult for further information about any one of these careers.

The author portrays these "medical occupations" in narrative form, depicting "Joe," just out of high school, seeking advice from a prominent member of each of these professions, who intelligently divulges from his experiences precisely what a young man wants to know before taking this very important step of his life—the choosing of a vocation. An example of the text is the day "Joe" spends with the local veterinarian, which takes him all the way from the humane treatment of an injured dog in an immaculate surgery to the vaccination of hogs in the average hog lot and the examination of a cow suffering from pneumonia, not to omit the inspection of viscera at the municipal (?) abattoir, in all, a busy day showing that the author *did* study the details of his subject before going to print. Klinefelter's little tome is a gripping story, well told, that certainly belongs in the library of every high school and college presuming to prepare young men for the mundane whirl.

PRACTICAL IDENTIFICATION OF ENDO-PARASITES FOR VETERINARIANS. John H. Whitlock, Assistant Professor of Veterinary Pathology, Kansas State College. 37 pages, with 30 line drawings. Burgess Publishing Co., Minneapolis, Minn. Price, $1.25.

The control of internal parasitism of domestic mammals can progress successfully only when those so engaged are able to identify, by groups or individually, the hundreds of parasitic species now known to be invaders. This is no easy task for the man in the field. Of course, he may submit specimens to his state or federal diagnostic laboratory; and should do so, especially when he encounters unusual species.

In preparing this publication, Dr. Whitlock has attempted to help the man in the field to help himself. To this end he has grouped the various parasites into keys, with an explanation for their use. Whenever possible, gross features are described for identification; but it should be understood that for many of the smaller forms, microscopic characteristics must be relied upon. Scientific names are listed accurately and common names are included, if such exist.

Parasite-identifying keys have long been used by the specialist. This publication makes available such a method to those whose interest in the subject is broader.

E. A. B.

A mongrel dog that recently bit twenty residents of south-side Chicago was infected with rabies, it was discovered. After his reign of terror, the animal was taken to the city pound. Euthanasia was performed and postmortem examination revealed the presence of the disease.

THE NEWS

OF GENERAL INTEREST

Professional Club at the New York Fair

The Professional Club, an incorporated organization, will be a feature of special interest to veterinarians who visit the New York World's Fair. For the first time at any international exposition, professional public health workers will have an opportunity to meet one another in club quarters reserved exclusively for their use. In congenial, luxurious surroundings, club members may renew old acquaintances with professional colleagues from the world over and discuss with representatives of ethical manufacturers the newest scientific developments of these companies in the cure, care and prevention of disease.

The club is to occupy an area of 5,000 square feet on the main floor of the exhibit building, with a main entrance from the garden between the science and medical wings of the building, and another from the Hall of Man. Its lounge is a wide, kidney-shaped chamber, ultra-modern in its decoration and furnishings, and equipped with a bar. Around the long arc of the lounge there are to be glassed-in cases in which sponsors of exhibits in the Hall of Medical Science may make displays of their products.

Professional memberships will carry no dues, and the facilities of the club will be open only to professional members and accredited representatives of participating organizations whose financial support made possible the educational exhibits in the building.

Kennel Club to Study Rabies Problem

On May 2, the American Kennel Club, 221 Fourth Ave., New York City, will convene. A committee of experts they have chosen will discuss the problem of rabies as it affects the dog and mankind. The personnel of this committee are: Walter Arnold, dog breeder and judge, Westfield, N. J.; Dr. Edwin Reginald Blamey, official veterinarian of the American Kennel Club; Arnold J. Brock, of the law firm of Goldsmith, Jackson & Brock, New York City; Harry I. Caesar, director of the American Kennel Club; Dr. J. E. Gordon, professor of preventive medicine and epidemiology, Harvard University Medical School, Boston, Mass.; Dr. Roscoe R. Hyde, professor of immunology, Johns Hopkins School of Hygiene, Baltimore, Md.; Dr. Charles J. McAnulty, former president of the New Jersey Veterinary Medical Association, Ventnor, N. J.; Dr. L. A. Merillat, executive secretary, American Veterinary Medical Association, Chicago, Ill.; William L. Smalley, director of the American Kennel Club, Chairman.

Although not a member of the committee, Dr. Leslie T. Webster of the Rockefeller Institute for Medical Research, New York City, has consented to attend the meetings and participate in the discussions. Dr. John L. Rice, commissioner of health of New York City, has also offered his assistance.

Revised A. V. M. A. Publicity Plans

Plans for the 1939 publicity of the A.V.M.A. have been somewhat revised, and the complete plan with the proposed changes has been submitted for the approval of the Executive Board.

As outlined, the campaign will have three general objectives, which are: (1) To inform the general public about the high scientific standing and attainments of veterinarians and about the significance of their work in maintaining public health, and to urge animal owners to consult licensed graduate veterinarians about the illnesses of their animals; (2) to stimulate advance interest in the Memphis convention in order to have a large attendance and to obtain wide publicity for the meeting, and (3) to spotlight wide public attention upon the veterinary exhibit at the New York World's Fair.

In order to establish in the public's mind that licensed graduate veterinarians are outstanding scientists who should be consulted about all illnesses of animals, the following facts will be stressed: That the practice of veterinary medicine today is on a high scientific plane; that veterinarians put in five years of college study and training before they are licensed to practice or engage in food inspection; that veterinarians perform vital services in maintaining public health, through assuring wholesome foods free from disease, and that the veterinary profession is an economic necessity making possible the present system of animal husbandry. It will be pointed out that, through the efforts of veterinarians, countless

animals have been saved from early death and live healthier lives.

Radio talks, news stories and feature articles are being planned to carry these facts to the public. Emphasis will be placed upon the scientific achievements of veterinarians and the advances they have made in the care and treatment of animals.

News stories are being planned also, to be released locally, concerning the activities of men of the profession. Such stories should be helpful to the man locally and to the Association nationally.

The Memphis convention will receive strong publicity promotion during the months preceding it, in order to stimulate maximum attendance. News stories and radio talks are now being planned to spotlight interest on the events of the meeting. It is expected that these will attract the attention of editors so that the publicity total will equal, if not exceed, the record publicity total obtained for the Diamond Jubilee meeting in New York City last year.

The A.V.M.A. exhibit at the New York World's Fair undoubtedly will be of great value in acquainting the public with the valuable services of the veterinary profession. Every effort will be made to obtain as wide publicity as possible for this.

International Kennel Club Show

Below is a list of the breeds represented at the 38th annual show of the International Kennel Club which was held in Chicago in April. The total number of entries and the number for each breed are revealing figures on canine popularity.

Sporting Dogs (Gun)	
Cocker spaniels	118
English springer spaniels	39
English cocker spaniels	11
Irish water spaniels	4
Britanny spaniels	3
Clumber spaniel	1
Irish setters	40
English setters	36
Gordon setters	15
Pointers	17
German short haired pointers	4
Golden retrievers	11
Labrador retrievers	7
Chesapeake Bay retrievers	4
Total	310
Sporting Dogs (Hound)	
Dachshund	41
Beagles	20
Basset hounds	10
Borzois	8
Irish wolfhounds	4
Afghan hounds	3
Norwegian elkhound	1
Bloodhounds	1
Total	88
Nonsporting Dogs	
Boston terriers	58
Schipperkes	48
Chow chows	37
Bulldogs	26
Dalmatians	24
Poodles	10
French bulldogs	6
Keeshonden	5
Total	214
Working Dogs	
Doberman Pinschers	65
Collies	55
German shepherd dogs	32
Great Danes	22
Boxers	14
St. Bernards	11
Samoyedes	10
Shetland Sheepdogs	10
Old English sheepdogs	6
Newfoundlands	3
Welsh Corgis (Pembroke)	3
Giant Schnauser	1
Rottweiler	1
Total	233
Terriers	
Scottish terriers	41
Wire haired fox terriers	39
Kerry blue terriers	31
Irish terriers	21
Airedale terriers	20
Welsh terriers	18
Sealyham terriers	17
Standard Schnauzers	16
Bull terriers	16
Smooth fox terriers	14
Bedlington terriers	11
Skye terriers	9
Staffordshire terriers	8
Miniature Schnauzers	7
West Highland White terriers	7
Dandie Dinmont terriers	7
Cairn terriers	5
Manchester terriers	1
Total	288
Toys	
Pomeranians	28
Pekingese	26
Miniature Pinschers	16
Chihuahuas	15
Toy Manchester terriers	13
Pugs	6
Yorkshire terriers	6
Japanese spaniels	5
Papillons	5
Affenpinschers	4
Brussels Griffons	2
Total	126
Miscellaneous	5
Grand total	1,259

Though this roster of dog breeds is by no means complete, it does give an insight to the complexity of small animal practice. It does not require the wisdom of a naturalist to see more mental, physical and occupational diversity in this file of canine purebreds than between the various species (not breeds) of farm live stock, not excluding the difference between the cackling hen and the sturdy herd bull. The variety of owners the list represents is the theme for another thesis.

Contributions to World's Fair Exhibit

John R. Mohler reports the following contributions to the fund being raised to finance the Association's exhibit at the New York exposition: Ray Gaskill of Pembina, N. Dak.—$10; Long Island Veterinary Medical Association—$50; Ohio State Veterinary Medical Association—$75; and Pennsylvania State Veterinary Medical Association—$500.

These contributions bring the total to $3,058.20.

COMING MEETINGS

Small Animal Hospital Association, Los Angeles, Calif. May 2, 1939. R. W. Gerry, Secretary, 8474 Melrose Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, New York City. May 3, 1939. C. R. Schroeder, Secretary, New York Zoölogical Park, Bronx, N. Y.

Connecticut Veterinary Medical Association. G. Leroy Cheney's Hospital, Woodbridge, Conn. May 3, 1939. George E. Corwin, Secretary, State Office Building, Hartford, Conn.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. May 4, 1939. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. May 4, 1939. W. T. Hufnall, Secretary, 1612-14 E. Alabama Ave., Houston, Texas.

Ak-Sar-Ben Veterinary Medical Association. Omaha, Neb. May 8, 1939. J. D. Ray, Secretary, 1124 Harney St., Omaha, Neb.

Willamette Valley Veterinary Medical Association. Albany, Ore. May 8, 1939. T. Robert Phelps, Secretary, 1514 Washington St., Oregon City, Ore.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. May 9, 1939. W. A. Young, Secretary, 157 W. Grand Ave., Chicago, Ill.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. May 10, 1939. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building 3919 John R St., Detroit, Mich. May 10, 1939. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Kansas City Veterinary Medical Association. Kansas City, Mo. May 15, 1939. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. May 15, 1939. Glenn A. Tucker, Secretary, Vista, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. May 17, 1939. Charles Eastman, Secretary, 725 S. Vancouver Ave., Los Angeles, Calif.

Massachusetts Veterinary Association. Hotel Westminster, Copley Square, Boston, Mass. May 24, 1939. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. May 24, 1939. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Oklahoma Veterinary Medical Association. Artesian Hotel, Sulphur, Okla. June 5-6, 1939. F. Y. S. Moore, Secretary, McAlester, Okla.

Texas, State Veterinary Medical Association of, and Texas A. & M. College Short Course for Veterinarians. Texas A. & M. College, School of Veterinary Medicine, College Station, Texas. June 6-7, 1939. M. B. Starnes, Corresponding Secretary, 202 City Hall Annex, Dallas, Texas.

Ohio State University Veterinary Conference. Ohio State University, Columbus, Ohio. June 14-16, 1939. O. V. Brumley, Dean, Ohio State University, Columbus, Ohio.

Missouri Veterinary Medical Association. Saint Louis, Mo. June 15-16, 1939. C. L. Campbell, Secretary, 7224 Tulane St., Saint Louis, Mo.

American Association for the Advancement of Science. Milwaukee, Wis. June 19-24, 1939. F. R. Moulton, Secretary, Smithsonian Institution Building, Washington, D. C.

Ninth Annual All-Day Practitioner's Clinic. Dairy Cattle Congress Grounds, Waterloo, Iowa. June 20, 1939. Sponsored by the Eastern Iowa Veterinary Association. C. E. Juhl, Chairman, Osage, Iowa.

California State Veterinary Medical Association. Santa Barbara, Calif. June 26-28, 1939. Chas. J. Parshall, Secretary, 319 B St., Petaluma, Calif.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. June 27-29, 1939. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

North Carolina State Veterinary Medical Association. Charlotte, N. Car. June 28-29, 1939. P. C. McLain, Secretary, Route 4, Charlotte, N. Car.

Northwest Veterinary Medical Association. New Washington Hotel, Seattle, Wash. July 10-12, 1939. V. C. Pauhlman, Secretary, 1524 Fifth St., Chehalis, Wash.

World's Poultry Congress, Seventh Annual. Cleveland, Ohio. July 28-August 7, 1939. W. R. Hinshaw, Chairman, Section on Pathology and Disease Control, University Farm, Davis, Calif.

American Veterinary Medical Association. Memphis, Tenn. August 28-September 1, 1939. L. A. Merillat, Executive Secretary, 221 N. La Salle St., Chicago, Ill.

State Board Examinations

Iowa Veterinary Medical Examining Board. June 13-14, 1939. All applicants must be in the office of the Division of Animal Industry not later than 8:00 a. m., June 13. Further information may be obtained from H. A. Seidell, Chief, Division of Animal Industry, State Capitol, Des Moines, Iowa.

Nebraska Board of Veterinary Examiners. June 26-27, 1939. Further information may be obtained from Mrs. Clark Perkins, Director, Bureau of Examining Boards, State Capitol Bldg., Lincoln, Neb.

(Continued on next page.)

U. S. GOVERNMENT

Regular Army Service

Lt. Colonel Horace S. Eakins is relieved from his present assignment and duty at Fort Benning, Ga., effective on or about May 15, 1939, and assigned to headquarters, Third Corps Area, Baltimore, Md., and in addition will act as attending veterinarian at Fort Howard, Md., and the Holabird Quartermaster Depot.

Lt. Colonel Frank H. Woodruff is relieved from duty at headquarters, Third Corps Area, Baltimore, Md., and additional duty as attending veterinarian at Fort Howard, Md., and Holabird Quartermaster Depot, Md., effective on or about May 15, 1939, and assigned to Fort Sill, Okla., for duty.

Lt. Colonel Charles O. Grace is relieved from duty at the Reno Quartermaster Depot, Fort Reno, Okla., is then assigned to Fort Meade, S. Dak., and will proceed to that station and report to the commanding officer for duty not later than May 15, 1939.

Lt. Colonel Edward M. Curley is relieved from assignment and duty at the United States Military Academy, West Point, N. Y., effective at such time as will enable him to comply with this order, and is then assigned to duty at Fort MacArthur, Calif. He will proceed at the proper time to New York, N. Y., and sail on the transport scheduled to leave that port on or about June 1, 1939, for San Francisco, Calif., and upon arrival, will proceed to Fort MacArthur and report for duty.

Lt. Colonel Elwood L. Nye is relieved from duty at Fort Meade, S. Dak., is then assigned to duty at the United States Military Academy, West Point, N. Y., and will proceed to that station and report to the Superintendent of the Military Academy not later than June 30, 1939, for duty.

Captain Charles S. Greer is relieved from duty at the Robinson Quartermaster Depot, Fort Robinson, Neb., effective on or about May 1, 1939, is then assigned to station at Fort Riley, Kansas; will proceed to Fort Des Moines, Iowa, and report to the commanding officer for temporary duty. At the proper time he will stand relieved from such temporary duty and will proceed to Fort Riley, and report to the commandant, the Cavalry School, on September 10, 1939, for duty as a student in the 1939-40 regular course.

Captain Bernard F. Trum is relieved from assignment and duty as a student at the University of Kentucky, Lexington, Ky., and additional duty at the Lexington Remount Area, effective upon completion of his present course of instruction on or about June 1, 1939, and assigned to the Robinson Quartermaster Depot, Fort Robinson, Neb., for duty.

Captain Daniel S. Stevenson is assigned to duty at the Reno Quartermaster Depot, Fort Reno, Okla., upon completion of his present tour of foreign service in the Philippine Department.

The War Department has announced a competitive examination to be held from July 24 to July 29, 1939, both dates inclusive, for the purpose of qualifying Doctors of Veterinary Medicine for appointment as First Lieutenant in the Veterinary Corps of the United States Army to fill contemplated vacancies.

Graduates of recognized veterinary colleges are eligible for the examination provided they are not less than 22 9/12 years of age at the time of examination and will not be over 32 years of age at the time it is possible to tender them a commission.

Applicants will be authorized to appear before examining boards convened at Army stations in representative sections of the United States to conduct the examination.

Complete information and application blank will be furnished any interested veterinarian upon request to The Adjutant General, War Department, Washington, D. C.

Applications for this examination will not be considered if received after July 1, 1939.

Veterinary Corps Reserve

NEW ACCEPTANCES

(First Lieutenants)

Cyrog, Robert John, c/o Christensen Animal Inst., 730 Hibbard Rd., Wilmette, Ill.

(*Continued from page* 555)

Connecticut State Board of Veterinary Registration and Examination. The next examination will be held in the office of the secretary, room 285, State Office Building, Hartford, July 5, 1939. Further information may be obtained from Geo. E. Corwin, Secretary and Treasurer, State Office Building, Hartford, Conn.

Illinois State Board of Veterinary Examiners. Examinations will be held in Chicago, July 24-25, 1939. Applications must be filed on blanks furnished by the Department of Registration and Education, Springfield, not later than 20 days before that date. The Board consists of W. W. Warnock, Aledo; W. H. Shaw, Pawnee, and L. A. Merillat, Chicago, Chairman. The fee is $20.00.

Donelson, Henry, 503 U. S. Court House, Fort Worth, Texas.

Eversole, Gardner Staples, P. O. Box 125, East Lansing, Mich.

Moore, Rufus Oliver Jr., 2217 7th St., Tuscaloosa, Ala.

Wilson, Neil Oliver, 1708 S. Meridian Ave., Alhambra, Calif.

PROMOTIONS

To Captain: Walter Edward Dicke, 137 Grant St., Carthage, Mo.

To Captain: Wm. Robert Kermen, 1436 N. Formosa Ave., Hollywood, Calif.

To 1st Lieut.: Emanuel Tarlow, 2219 Westchester Ave., New York, N. Y.

Disposal of Army Mules and Horses

One of the problems confronting the federal government which seems to be regarded as important enough to make the headlines of the metropolitan newspapers is the bill before Congress proposing to set down rules for the disposition of army mules and horses when they have served their purposes. Sentimental friends of the horse and mule appear to want Congress to appropriate money for keeping such animals in retirement until they die a natural death, preferring this to killing the suffering unfit or to selling the better ones where they can still serve a useful purpose.

Radio Broadcast on Equine Sleeping Sickness

On April 14, 1939, the United States Department of Agriculture sponsored a radio broadcast on sleeping sickness of horses and mules over an extensive network of stations as a feature of the National Farm and Home Hour. The broadcast was in the form of a dialogue between Dr. John R. Mohler, Chief of the Bureau of Animal Industry, and Wallace Kadderly, in charge of the Department's Radio Service. The discussion reviewed the status of knowledge concerning the disease to date.

Dr. Mohler expressed the belief that a new chick-embryo vaccine will be largely instrumental in offsetting future losses and in reducing the threat of encephalomyelitis to the horse and mule industry of the country.

New U. S. Order Aids Live Stock Disease Control

Still further health protection for domestic animals of the United States, already closely guarded in this respect, is provided by a new regulation of the U. S. Department of Agriculture. Designated as Amendment 14 to Bureau of Animal Industry Order 276, the new provision requires a special permit for importing cultures or collections of organisms or viruses and for transporting them from state to state. It requires a special permit also for the similar handling of experimental animals treated or inoculated with such materials.

The amendment became effective March 15, 1939, through order of the Secretary of Agriculture, who issued it under authority vested in him by Congress. Administration of the new regulation will be supervised by Dr. D. I. Skidmore, Chief of the Division of Virus-Serum Control of the Bureau of Animal Industry. Doctor Skidmore points out that the new safeguard, while giving additional protection to live stock health, will not interfere with the normal importation or interstate movement of veterinary biological products as customarily handled under regular permits.

From time to time cultures of organisms and viruses that are regarded as dangerous to the health of live stock in the United States have been offered for admission from abroad. In some cases the causative agents of animal diseases not existent here have been brought into this country because there was no specific control of such organisms or carriers of them.

The provisions of the amendment apply to all viruses, bacteria, living organisms, and the like from foreign countries and to all experimental animals such as mice, pigeons, guinea pigs, rats, ferrets, and rabbits which have been treated or inoculated with such viruses and organisms. They apply also to animals which are diseased or infected with any disease or which have been exposed to any such infection.

B. A. I. History Presented to National Archives

In response to a request recently received from an official of the National Archives, the U. S. Bureau of Animal Industry has presented that branch of the government with a copy of the bound volume, "The Bureau of Animal Industry of the U. S. Department of Agriculture, Its Establishment, Achievements, and Current Activities," prepared in 1924 by the late Dr. U. G. Houck and other members of the Bureau's staff. This book, of 390 pages, is the most comprehensive existing account of the first 40 years of B. A. I. achievements.

The National Archives Building, recently constructed in Washington, D. C., was built especially for the safe-keeping and preservation of valuable government documents. The building is said to be not only fireproof and burglarproof, but also earthquake-resistant and as time-proof as modern engineering skill has been able to build.

The reserve supply of the book has been exhausted for several years and copies now held privately are expected to become increasingly rare with the passing of time. Presentation of the book to the National Archives insures that the valuable information contained will be available to posterity.

AMONG THE STATES

Secretaries of state associations, resident state secretaries, foreign corresponding secretaries, secretaries of local associations and members in general are requested to keep this department supplied with information that will portray the veterinary situation of the states, provinces and countries within the jurisdiction of the Association. The object is to make this a complete report of what is transpiring in the veterinary profession. It is up to you!

California

Chances for a college of veterinary science at the University of California, Berkeley, seem very slim. On April 6, the universities and colleges committee of the state senate turned over the veterinary college bill to the finance committee, with a recommendation that it be rejected. It seems that the legislators approved of the principle, but decided that the $500,000 required for the project is too great an amount, at least for the time.

District of Columbia

During the coming months, John R. Mohler, Chief of the U. S. Bureau of Animal Industry, will address the Seventh World's Poultry Congress at Cleveland, Ohio, the Third International Veterinary Congress on Microbiology, and the Regional Laboratory at East Lansing, Mich., besides addressing the Association at Memphis. H. W. Schoening of the Bureau will speak on equine encephalomyelitis at the Memphis meeting.

Connecticut

George E. Corwin, deputy commissioner on domestic animals, has been reappointed by Governor Baldwin to the State Board of Veterinary Registration and Examination, for a five-year term beginning July 1, 1939. Dr. Corwin is secretary of the Board. Also appointed to the Board was E. M. Bitgood of Middletown. He will fill the vacancy caused by the death of Peter T. Keeley of Waterbury.

The WPA project now continued as project 3202 and sponsored by the Commission on Domestic Animals and the Dairy and Food Commission, which was set up in October, 1935, for the purpose of detecting mastitis in dairy cows by means of the bromthymol test, has to its credit the testing of 13,805 herds containing 164,448 cows, as of March, 1939. The incidence of mastitis was slightly over 20 per cent.

Illinois

R. E. McDaniels, coördinating bacteriologist of the State of Illinois, discussed the various phases of virus diseases before the Chicago Veterinary Medical Association, April 11. The Chicago association holds regular monthly meetings, with the exception of two vacation months in the summer. This it has done without interruption since 1896.

The state association is considering the introduction to the state legislature of a bill to amend the Veterinary Practice Act of 1889.

Iowa

A. H. Quin, editor of the Fort Dodge Bio-Chemic Review, reports that the North Central Iowa association had the largest attendance of any local association meeting ever held in the United States.

Right now, plans are being made for the Ninth All-Day Practitioners' Clinic to be held in Waterloo, in June.

W. J. Pirie, secretary of the Advisory Committee of the Eastern Iowa association, has informed the central office of a letter he wrote, in behalf of the Eastern Iowa group, to J. D. Harper, editor of the *National Live Stock Producer*, complimenting him on the fine editorial which appeared in the April number of that publication. Here is the editorial, entitled "Live Stock Health," that inspired Dr. Pirie's letter:

> An increasing amount of light has been shed annually on human health through a study of animal diseases. In fact, many diseases in animals are transmitted to humans. Veterinary research work has, from the earliest times, made important contributions to human medicine. Live stock producers have a two-fold interest, therefore, in this subject and those who visit the New York World's Fair will be interested in an exhibit, "Veterinary Science and Public Health."
>
> The veterinarian has an increasingly large field of service because of the new diseases that are of animal origin, or may attack either man or beast.

Massachusetts

In part I of the April issue of the JOURNAL, in the report of the Committee on Education, the following statement concerning Middlesex University, Waltham, appeared:

> There is one private veterinary school starting at Middlesex University in Massachusetts. There is one veterinarian there at present. They may have more in the course of time. I am not saying this disparagingly at all. They have supplied us with data as requested, but whether the number of prospective students is sufficient to warrant a commercial venture in that line, I am not able to state.

Believing that this statement might be misinterpreted by some of the readers, C. Ruggles Smith, president of the University, has requested that the following communication be published:

> Middlesex University is an eleemosynary, tax-exempt, non-profit, educational institution, chartered by a special legislative act by

the Commonwealth, said act having been approved and recommended by the state department of education. It was specifically authorized to establish a school of veterinary medicine in order to meet the demand for veterinary training in this area.

The establishment of this new department is in no conceivable sense of the word a "commercial venture," since under the Massachusetts law and under the terms of our charter it would be a criminal offense for any individual to derive a profit from the institution. Middlesex University is a "private" institution only in the sense that Harvard, Yale and Columbia are "private" institutions, that is, none is supported by public funds.

We have submitted to your Committee on Education complete information concerning our pre-veterinary and veterinary faculty, and we feel that the publication at this time of the sentence, "There is one veterinarian there at present." would also tend to create an unfair impression.

The trustees of Middlesex University have recently approved the erection of a new building for the School of Veterinary Medicine, to cost $100,000. Work has already started on this structure, after Drs. E. T. Hallman and W. A. Hagan of your Committee on Education were kind enough to examine the original plans and suggest certain changes.

Michigan

E. C. W. Schubel of Blissfield, Mich., writes:

Elmer Beamer of this city has been appointed Commissioner of Agriculture to succeed John B. Strange of Grand Lodge. The importance of this is that Mr. Beamer is one of the most friendly men in the live stock industry and a great admirer of the veterinary profession. Besides being a dirt farmer, he is well known throughout the country for his expertness in marketing farm products.

Nevada

As a result of an examination held at Reno on February 10, 1939, the Nevada Board of Veterinary Medical Examiners has issued licenses to practice in the state to the following:

E. Thompson Martin (K. C. V. C. '14), formerly of Tulsa, Okla. now residing at 30 California Ave., Reno.

Howard F. Fleming (Corn. '18) of Gardiner, N. Y.

George Miner Wilson (Iowa '37), CCC Headquarters, Seventh and "K" Streets, Sacramento, Calif.

Edmund B. McCormick (K. S. C. '37), CCC Headquarters, Seventh and "K" Streets, Sacramento, Calif.

New Jersey

At the invitation of John T. McGrann of Trenton, about 20 veterinarians of the vicinity of that city met on March 23 to establish a local association. Another meeting is scheduled for April 27, at which time a name for the society will be selected and other important matters will be decided. C. B. Johnson of Trenton, secretary, will select at each session three new committee members to supervise the activities of the ensuing meeting. As there is no provision for a permanent presiding officer, this committee will in turn select one of its members to act as chairman.

New Mexico

Under the guidance of the resident state secretary, T. I. Means, membership in this state promises to be 100 per cent within a relatively short time.

Oklahoma

The state legislature did not pass the Bang's Disease Control Bill, it having failed to make the necessary appropriation, notwithstanding that the state stands third in the number of cattle tested for Bang's disease.

Except for the usual quantity of hog cholera and swine erysipelas, the animal disease situation is satisfactory. Secretary Douglas reports that cases of equine encephalomyelitis already have been reported from widely separated sections, indicating that an early outbreak can be expected this year.

Oklahoma offers several good openings for general practice, and many promising young practitioners are now locating there.

Pennsylvania

The General Magazine and Historical Chronicle, a quarterly issued by the University of Pennsylvania, shows on the cover of the April, 1939, issue that it was "Printed and Sold by B. Franklin in 1741."

South Carolina

In the *Chester Reporter*, March 30, a quack advertised his services in vaccinating horses against blind staggers and offered treatment for influenza, shipping fever, distemper and pink eye. He also advertised "a fine colic remedy," and listed about 25 other maladies which he claims he can treat.

A new bill designed to regulate further the practice of veterinary medicine and surgery is now before the state legislature.

Tennessee

During the month of March, 101 outbreaks of hog cholera were reported from 29 counties in the state. Most seriously affected were Hamblen County, with 17 outbreaks, and Dickson and Wayne counties, with 12 and 11, respectively. A total of 38 outbreaks of erysipelas occurred in 16 counties, and four outbreaks of hemorrhagic septicemia were reported from three counties. Each of five counties listed one outbreak of blackleg.

The Committee on Local Arrangements for the Memphis convention meets once a month and sends detailed reports of their activities to the central office.

J. L. Arnandez, chairman of the Publicity Committee, writes that the firm of McCallum & Robinson, rug manufacturers, will play a big part in the entertainment of the ladies during the convention.

MEETING REPORTS

1939 Veterinary Congress, Philippine Veterinary Medical Association

The 1939 Veterinary Congress, under the auspices of the Philippine Veterinary Medical Association, was held February 20-22 at the assembly hall of the College of Veterinary Science, Bureau of Animal Industry grounds, Pandacan, Manila, P. I.

The opening address was delivered by Teodulo Topacio, president of the Association and chief of the Veterinary Research Division, Bureal of Animal Industry, Manila. Following, Gregorio San Augustin, director of the Bureau of Animal Industry and dean of the College of Veterinary Science, University of the Philippines, introduced Hon. Juan J. Ledesma, former delegate to the Constitutional Convention, who addressed the group.

Many instructive clinical demonstrations and a host of fine scientific papers were given during the three-day session. A highlight of the activities was the banquet held on the evening of the third day at the Philippine Columbian Association Building. Hon. Benigno Aquino, Secretary of Agriculture and Commerce, was the guest of honor on this occasion.

Long Island Veterinary Medical Association

The February meeting of the Long Island Veterinary Medical Association was held at the Shadow Lawn, Hempstead, Long Island, N. Y., on Thursday evening, February 23.

The annual election of officers was held, with the following being chosen: Oscar Glueck of Blue Point, L. I., president; Lawrence T. Waitz of Hempstead, L. I., vice-president; and Herman Tax of Farmingdale, L. I., secretary-treasurer.

Dr. Craig of the University of Pennsylvania spoke on "Some Practical Diagnostic Methods."

March Meeting

The March meeting of the Association was held at the Stirrup Cup Castle at Oakdale, Long Island, on Wednesday evening, March 29.

M. A. Emmerson, assistant professor of veterinary surgery and obstetrics at the University of Pennsylvania, spoke on "X-Ray Therapy of Domestic Animals." He made specific reference to the results that were obtained by the application of x-ray therapy in the treatment of the following: cow with actinomycosis; gelding with multiple tumors of the right mandible, commissure of the mouth and cheek; horse with canker in four feet; horse with canker in three feet; collie with carcinoma of the paw; fox terrier with epidermal parapractocarcinoma; Irish Setter with a sarcoma posterior to the ear; Great Dane with osteogenic sarcoma in distal end of the radius; and a dog with epidermal carcinoma of the paw.

April Meeting

This meeting was held on Thursday, April 20, at Port Jefferson, Long Island. A detailed report will be given later.

Herman Tax, *Secretary.*

Northwest Indiana Veterinary Association

G. W. Andree of Rensselaer was elected president of the Northwest Indiana Veterinary Association at a meeting held at the Lunghi restaurant, Rensselaer, on the evening of March 16. Harvey Smith of Crown Point was chosen vice-president, and L. E. Andres of Remington was reëlected secretary-treasurer. Included in the program were motion pictures on equine encephalomyelitis.

Southwestern Minnesota Veterinary Medical Association

The 19th semi-annual meeting of the Association was held in Sleepy Eye, Minn., March 21, 1939. The session was opened with a banquet at the Congregational Church and was served by the ladies of the church. After the banquet, the ladies who attended were entertained by Mrs. E. G. Hughes and Mrs. W. A. Anderson at the home of Dr. and Mrs. Hughes.

The speaker of the evening was Mr. Ruben Hovland, senior veterinary student at Iowa State College, Ames, Iowa, who presented a paper on "Some of the More Interesting Cases Handled at the Clinic at Iowa State College."

Officers for the ensuing year were elected as follows: C. L. Tompkins of Redwood Falls, president; E. H. Enge of Comfrey, vice-president; L. E. Stanton of Jackson, secretary-treasurer; and I. M. Ford of Edgerton, member of the Board of Trustees.

A committee report by Dr. Van Duzee regarding the vaccination of horses for encephalomyelitis produced considerable discussion. A resolution was passed that the Association go on record as disapproving of the practice, of some commercial houses, of establishing distributing depots for veterinary biologics in drug stores.

L. E. Stanton, *Secretary.*

PERSONAL NOTES

Marriage

RAY LEWIS (O. S. U. '38) of Sidney, Ill., to Miss Imogene Chambliss, that city, March 3, 1939.

Activities

G. T. WOODWARD (Wash. '24), has resigned from his position with the U. S. B. A. I. and relocated for practice at Fallon, Nev.

E. A. BORTMAN (Mich. '37), who practiced in Detroit, Mich., for three years, has opened an office for general practice in Utica, Mich.

CHARLES W. FOX, who was graduated from Ohio State University in March, plans to open an office in Sycamore, Ohio, for general practice.

W. W. DIMOCK (Corn. '05) of the University of Kentucky, Lexington, Ky., who was stricken with pneumonia recently, is reported to be recovering.

W. B. BRADLEY (Mich. '38) has entered private practice at Caledonia, Mich. Previously he was associated with the Pet Milk Co. at Greensboro, Md.

W. C. HIRSCHEY (Mich. '38) of Albion, Mich., has returned from an extended stay in Castorland, N. Y., his former home, where he recuperated from an operation.

V. A. YOUNT (Colo. '36) has resigned from the U. S. B. A. I. meat inspection force at Kansas City, Mo., and is now located in Muskogee, Okla., for general practice.

JAMES B. CORCORAN (Colo. '33), formerly with the U. S. B. A. I. on meat inspection at Indianapolis, Ind., has resigned from the service and is now associated with H. W. Ayers (Ont. '08) in general practice at Oklahoma City, Okla.

W. B. MASSIE (Mich. '16) of Boston, Ind., was the principal speaker at the annual banquet of the Preble County (Ohio) Horsebreeders' Association, held in Eaton, Ohio, recently. His topic was "Sleeping Sickness in Horses."

F. C. TUCKER (McK. '09), prominent poultry practitioner of Claypool, Ind., is one of the leaders in the movement to stimulate advance interest in the World's Poultry Congress, to be held in Cleveland, Ohio, July 28 to August 7.

C. E. TURNBULL (Gr. '11) recently constructed a small animal hospital in Dearborn, Mich., which has attracted the attention of every person for miles around. The building, ultra-modern in every respect, was built according to Dr. Turnbull's own specifications at a cost of $12,000.

L. B. FORD (Ont. '16) of Swanton, Ohio, suffered a broken and severely lacerated nose while treating a steer on a neighboring farm. As Dr. Ford was administering a hypodermic to the animal's shoulder, the steer jerked his head in Dr. Ford's direction and the short horn caught him squarely on the nose.

E. S. WEISNER (Mich. '37) is conducting a series of lessons in poultry diseases at the Centreville high school, Burr Oak, Mich. As part of the course, Dr. Weisner, who specializes in poultry disease work, diagnoses the ills of any sick birds which students may bring to the class. The fifth lesson in this series was given on March 21.

ROBINSON W. SMITH (Chi. '04), veterinarian well known from coast to coast, was elected mayor of his city, Laconia, N. H., at the recent April election. Mayor Smith won the election on the fine New England policy, "Pay as you go." In the veterinary profession east of the Alleghenies, Mayor Smith is best known as the former president of the United States Livestock Sanitary Association.

STANTON YOUNGBERG (O. S. U. '07) of Georgesville, Ohio, is now in Manilla, P. I., where he is studying the prospects of a settlement for refugees of central Europe. Dr. Youngberg is working in the Philippines with a group known as the Mindanao Exploration Commission, which has been delegated by the Refugee Economic Corporation of New York City to survey the opportunities for settlement of refugees on the Islands. Mindanao is the island being considered at present.

DEATHS

William R. Swan of Stevens Point, Wis., died at the age of 54 on March 18, 1939. He had been in poor health since suffering serious injuries resulting from an automobile accident in December of 1937. His condition became critical following a stroke, and he was removed to Saint Michael's Hospital, where he died two days later.

Dr. Swan was born on April 14, 1885, in Belmont, Wis. After graduating from the Chicago Veterinary College in 1908, he entered private practice at Stevens Point, and was active professionally there until the time of his accident. Dr. Swan joined the national association in 1916. He was an active member and president for many years of the Wisconsin Veterinary Medical Association and also belonged to the Central Wisconsin association.

J. S. H.

Robert T. White, veteran executive of the Corn States Serum Co., passed away at his home in Cedar Rapids, Iowa, March 22, 1939, after an illness of nine weeks.

Dr. White was born in Kewanee, Ill., on June 14, 1860. He practiced in Annawan, Ill., until the death of his wife in 1915. Shortly after, he became sales manager of Corn States' Cedar Rapids branch, where he built up a splendid record of achievement for his firm and himself.

William J. Selkin of Bronx, New York, died on March 23 after undergoing a major operation three days earlier.

He was born in Albany, N. Y., on November 21, 1891. After graduating from Cornell University in 1913, Dr. Selkin entered the service of the U. S. Bureau of Animal Industry and was stationed in Alabama in charge of tick eradication. Later he was transferred to the Jersey City stock yards as antemortem inspector. About eleven years ago he joined the New York City Health Department as veterinarian in the food division (meat and poultry inspection). Dr. Selkin also was engaged in private practice and maintained an animal hospital at 604 E. Gun Hill Road, Bronx, New York. He joined the A. M. V. A. in 1937.

C. R. S.

M. O. Anderson of Orenco, Ore., died on March 18, 1939.

He was born at York, Ontario, November 2, 1857. After graduating from the Ontario Veterinary College, class of 1886, he was engaged in private practice for several years. In March of 1898 he entered the service of the U. S. Bureau of Animal Industry. During the years before his retirement from the Bureau in 1930, Dr. Anderson served as inspector- in-charge at Austin, Minn.; Ottumwa, Iowa; South Saint Joseph, Mo.; South Saint Paul, Minn.; and Portland, Ore. He was a member of the A. V. M. A. from 1915 until 1931.

E. E. C.

Thomas O. Scott died on March 30, 1939, while working at his hospital in Waco, Texas. He had been in poor health for quite some time.

Born in Jasper, Texas, October 30, 1883, Dr. Scott attended the Kansas City Veterinary College, from which he was graduated in 1912. He served overseas during the World War and was a past commander of the American Legion Post in Waco. He was a Knight Templar, Mason, and Shriner. He was a member of the Texas Veterinary Medical Association, and had held the offices of vice-president and president. While serving as president of the Association, he obtained a policy arrangement with the Agricultural Extension Department of his state which was later adopted as a resolution by the A. V. M. A. Dr. Scott joined the national association in 1919.

Norman A. Buttle, formerly president and vice-president and, since 1934, treasurer of the Winthrop Chemical Company, died on April 17, 1939.

One of the original founders of the company, Mr. Buttle had proved himself a loyal, earnest and indefatigable executive, and his high ability accounted substantially for the progress of the firm since its establishment in 1919.

Harry A. Meisner of Towson, Md., died on March 11, 1939.

Born in East Baltimore, Md., Dr. Meisner was graduated from the University of Pennsylvania in 1890. Before establishing the practice he maintained in Towson, he had owned and operated an animal hospital for 35 years in Baltimore. He was credited with being the only man in Baltimore County who had ever performed a successful operation on a Bengal tiger, using only a local anesthetic. Dr. Meisner joined the national association in 1891.

Hugo Cornehl passed away at his home in Detroit, Mich., on March 14, 1939, at the age of 69. He had been in poor health since September.

Born in Detroit, Mich., January 17, 1870, he graduated from the Grand Rapids Veterinary College in 1908. A few years later, he went to Carlsbad, Germany, to take further instruction in veterinary science. He entered the service of the Detroit Department of Health in 1919, where he served until retiring about two years ago. Dr. Cornehl joined the national association in 1923.

Lawrence Alexander Cottrell died suddenly at his home in Waterville, Minn., on March 4, 1939. Embolism, complicated from the effects of undulant fever, from which he had suffered for about a year, was given as the cause of death.

Dr. Cottrell had been associated with the Fort Dodge Laboratories since 1935, as manager of their Mankato, Minn. branch.

Henry N. Jeffries of Greenville, Ohio, died at the age of 80 on February 20, 1939. Born in Kentucky, Dr. Jeffries moved to Greensburg, Ind., at the age of seven years, with his parents. He was graduated from the Ontario Veterinary College in 1900. During the World War, he served as chief inspector for the U. S. Bureau of Animal Industry, at Kansas City, Mo.